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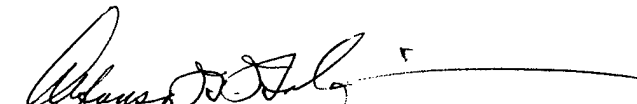
CO-MORBIDITY & MORTALITY: SIX DECADES
OF CARBON MONOXIDE POISONINGS

By

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25 SEP REC'D

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THESIS

Presented to the University of Texas

Health Science Center at Houston

School of Public Health

in Partial Fulfillment

of the Requirements

for the Degree of

MASTER OF PUBLIC HEALTH

**THE UNIVERSITY OF TEXAS HEALTH SCIENCE CENTER AT HOUSTON
SCHOOL OF PUBLIC HEALTH
Houston, Texas
August, 2002**

DEDICATION

To all the teachers and professors I've known and worked with over the past 37 school years. Chief among them of course is [REDACTED] -- my high school advanced biology teacher, my football coach, and my dad. During senior year he fanned the flames of scientific interest that he, himself, ignited in a curious kid a decade and a half earlier.

CO-MORBIDITY & MORTALITY: SIX DECADES OF CARBON MONOXIDE POISONINGS

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In Bexar County, Texas, 588 people died from carbon monoxide-related incidents between 1936 and 1995. Evaluation of these data reveals trends in both accidental and suicidal carbon monoxide fatality rates that dramatically increased to peak levels in the late 1950's and early 1960's, respectively, and then declined over the next few decades. By the five-year period 1991-1995, the overall rate of accidental carbon monoxide deaths in Bexar County had dropped to about half the rate experienced during 1936-1940. Decreases in suicidal carbon monoxide poisonings appear to have leveled off during the 1980's and 1990's at rates approximately one and one-half times their 1936-1940 level, and now account for three-quarters all the carbon monoxide deaths in the county. In general, during the final decade of study (1986-1995), males died from carbon monoxide five times as often as females. The suicide rate for non-Hispanic white males was nearly eight times higher than Hispanic males, and that of non-Hispanic white females was more than twelve times higher than Hispanic females. Deaths occurred among all age groups, with suicide rates exceeding accidental rates in all but the youngest and oldest groups. No carbon monoxide suicides occurred in children age 0-9, and among people 70 years of age or older carbon monoxide related suicides and accidental deaths occurred with equal frequency. While the overall trends in death rates appear reassuring compared to levels in the mid-twentieth century, further reductions in this ongoing health threat will likely require additional, coordinated efforts in public awareness, patient education, and medical research.

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I must also acknowledge the generosity of the National Institute of Child Health and Human Development, which funded the initial collection and tabulation of Bexar County death data for the period 1935 through 1985 as part of an original research project, entitled “Mortality Among Mexican Americans and Mexican Immigrants.” Data for the period 1986 to 1995 have been added periodically through the ongoing interest and energy of individuals at the Texas Department of Health and within the School of Public Health.

Finally, a special thanks to Beck and Em – not only for enduring countless hours at home while I was at the library or sitting at the computer, but more importantly for enduring countless hours listening to not-so-exciting stories about carbon monoxide.

Thesis submitted to the MPH Committee on July 5, 2002.

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BACKGROUND

Introduction

Carbon monoxide, a very simple molecule and a common product of incomplete combustion, is a familiar yet dangerously unfamiliar substance. To the average citizen it finds its way into discussions of automobile exhaust, suicides, and cigarette smoke. But misconceptions or lack of awareness seem to continue to cause hundreds, if not thousands, of accidental deaths in this country every year.^{1,2,3} This is, of course, in addition to an even greater number of intentional deaths, for which carbon monoxide is the chosen means of self-destruction. In fact, carbon monoxide remains the leading cause of all poisoning deaths in the United States (and, quite possibly, the world).^{4,5,6,7,8,9}

From a public health point of view, questions of public awareness and public safety remain the most important issues. Practically all of us sit in heavy traffic from time to time, the media carry occasional reports of families sickened or killed by faulty (or inappropriate) heating systems, many of us know of people who have intentionally died after starting cars in closed garages, and smokers are ever so discreetly warned that "*Cigarette smoke contains carbon monoxide.*" Due to its insidious properties, carbon monoxide gives little or no warning signs to unsuspecting victims. Lower level poisonings are frequently overlooked or misdiagnosed when patients tell their doctors about difficulties with headaches, dizziness, nausea, or weakness.^{6,10,11,12,13,14,15} Too often they receive treatment for viral- or food-related illnesses, then return home to the true source of the problem, only to have their symptoms persist for months... or worse.

Not surprisingly, media coverage of this important toxin tends to be limited to local stories about tragic household accidents, or to firemen overcome by smoke. This often translates into an underinformed (or sometimes misinformed) general public.^{16,17,18,19} On the other hand, volumes of data can be found in scientific journals about carbon monoxide uptake, its pathophysiology, blood levels found in emergency room patients, and noticeable changes in brain features of poisoning victims detected by magnetic-resonance imaging. Researchers have used animal models to chart in great detail the effects of carbon monoxide

on living systems, and much information has been compiled about the near- and long-term consequences of both acute and chronic exposure to this toxic gas. But important questions remain: What is really being done to bring local historical data and the collected scientific knowledge to the level of useful public understanding? Are there certain groups more at risk than others? And, should more be done about it?

The primary goal of this thesis is to review the health threats posed by carbon monoxide and to specifically relate these threats in a local context. Certainly an overview of carbon monoxide's chemical and physical characteristics, along with a review of its various production sources and a description of early signs and symptoms of carbon monoxide poisoning would be a worthwhile endeavor in itself. To better analyze the overall health burden of this "silent killer," however, a few other areas should be examined. For example, in Bexar County, Texas, approximately 600 people died from carbon monoxide-related incidents between 1935 and 1995, as recorded from death certificate data compiled from county records. These data will be presented, showing the distribution of deaths by manner of death, age, sex, and other demographics. This will help define carbon monoxide's role as a health concern in San Antonio and surrounding communities. Background information on chemical and physiological properties will be presented, along with a general review of some of the ongoing research efforts in the scientific community. Following the evaluation of the deaths experienced in Bexar County over the specified sixty-year period, comments will be offered and recommendations made relevant to the local threat characteristics found. This will include consideration of information currently available through the media and various public organizations. Ultimately, recommendations for targeted public health education in San Antonio and for additional local research will be possible. In addition, healthcare providers might find this study useful as a general review of carbon monoxide sources as well as the signs, symptoms, and sequelae of sub-lethal exposure.

Carbon Monoxide Sources and Basic Science

Each molecule of carbon monoxide (CO) consists of a single oxygen atom bonded to a carbon atom. This structure is extremely stable, and accounts for many of carbon

monoxide's chemical characteristics. In any combustion reaction, carbon is first oxidized to carbon monoxide. Further ("complete") oxidation to carbon dioxide requires an additional step with a much greater activation energy. But if sufficient time, heat, or oxygen are not available for the reaction to proceed, large amounts of carbon monoxide remain.²⁰ Such "incomplete" burning in fires, motor vehicle engines, industrial processes, cigarettes, and heating systems is probably the most readily apparent cause of carbon monoxide, and in fact man-made ("anthropogenic") sources account for about 85 million tons of American-made carbon monoxide each year.²¹ However, on a global scale, natural sources still produce the vast majority of carbon monoxide. Decomposition of plant and animal matter produces methane, whose reaction sequence with hydroxyl radicals in the atmosphere creates about 80 percent of the world's annual carbon monoxide production.²⁰ More than half of the remaining CO comes from other natural sources, including the breakdown of chlorophyll, and a wide variety of natural reactions in the oceans.²⁰ Of course, total production figures and natural "sinks" for carbon monoxide consumption are not especially important from a medical or public health point of view when one compares the relative distribution of the substance. For although anthropogenic sources produce less than ten percent of the world's carbon monoxide, approximately 98 percent of the carbon monoxide within US cities does result from human activities.²⁰ Clearly, these tiny pockets of high CO concentration, coinciding with our densest population areas, create the true problems of medical and public health concern.

Basic Physiological Issues

The majority of carbon monoxide found in the body is breathed directly into the lungs, although some amounts are introduced by other pathways. For example, skin absorption or inhalation of methylene chloride (a common component of paint removers and other solvents) produces CO when metabolized by the liver.²² The body's normal breakdown of hemoglobin and other heme-containing compounds also produces carbon monoxide, which accounts for small, but measurable, background levels of CO in the blood.²³ A colorless gas without taste or odor, carbon monoxide is similar to oxygen in molecular size, weight, and

diffusionary properties across various membranes, and so readily competes with oxygen for hemoglobin binding sites upon entering the lung capillaries. Inside the red blood cells, carbon monoxide binds with hemoglobin nearly as fast as oxygen does; once bound, however, its release rate is a mere fraction of that of oxygen. This “tighter binding” results in carbon monoxide having an overall net affinity for hemoglobin binding about 240 times that of oxygen under normal conditions.^{24,25,26} Studies have shown that although most carbon monoxide in the body is chemically bound to hemoglobin, ten to 15 percent is typically distributed to extravascular tissues where it is bound to other heme proteins, such as myoglobin (an oxygen storage protein), cytochromes (important in cellular respiration and detoxification pathways), and certain enzymes.²⁷ Because carbon monoxide is relatively insoluble in water, very little is physically dissolved in bodily fluids.

Since carbon monoxide’s most basic interactions involve hemoglobin, it is important to briefly review hemoglobin’s structure and function. Hemoglobin is a tetrameric protein (a “four-chain” protein). Each polypeptide chain surrounds and holds a relatively small organometallic heme group within its three-dimensional structure. An iron atom sits at the center of each heme group. These iron atoms each have a binding position available, intended for the temporary attachment of an oxygen molecule, thereby giving hemoglobin its oxygen-carrying ability. As in many biochemical reactions the attachment and release of oxygen at these sites at any given time depends on a number of variables, such as oxygen tension in the blood, availability of open binding sites, ambient temperature, and pH. As “designed,” oxygen is to be secured to the heme groups in the relatively oxygen-rich environment of the lungs, and then readily released to the tissues as the blood travels to oxygen-poorer sites around the body. A typical oxygen dissociation curve, showing percent of oxyhemoglobin saturation (or “oxygen binding ability”) as a function of blood oxygen tension, is represented by the graph in Appendix A. This figure demonstrates that as the surrounding level of oxygen is reduced, hemoglobin’s desire to hold onto oxygen drops, and so the oxygen being carried is released. The sigmoid shape of the curve is a result of “cooperative binding,” which means the binding of an oxygen molecule to one of the four hemoglobin subunits makes that hemoglobin molecule more likely to bind more oxygen at its

other heme groups. Portions of the hemoglobin molecule near the first oxygenated heme group shift slightly, which in turn alters the configuration of the molecule's four polypeptide units. These changes enhance the binding properties of the remaining heme groups, possibly by making the binding sites more accessible to additional oxygen molecules.²⁸ Binding of carbon monoxide to hemoglobin is characterized by a similar dissociation curve, but work done in the late 1980's at the University of Colorado showed that carbon monoxide exhibits an even greater binding cooperativity than oxygen, indicated by the steeper dissociation curve in Appendix B.²⁹ The findings that the oxygen and carbon monoxide curves are not exactly parallel altered long-held beliefs that the partitioning between carbon monoxide and oxygen binding to hemoglobin was constant at all levels of hemoglobin saturation. It is now known that carbon monoxide's affinity for hemoglobin ranges from about 170 to over 270 times that of oxygen as saturation increases.²⁹ In addition to successfully replacing oxygen in hemoglobin binding, carbon monoxide also shifts oxygen's dissociation curve to the left, as shown in Appendix C.^{30,31} This means that at a given blood oxygen tension, more oxygen will tend to stay bound to hemoglobin than if carbon monoxide were not present. This, of course, further aggravates carbon monoxide's toxic effect – not only is less oxygen given the opportunity to bind with hemoglobin, but that which does bind is not as readily released during delivery attempts to the oxygen-starved cells.

Another important comparison involves the different binding properties of hemoglobin and myoglobin. Myoglobin plays a crucial oxygen storage and transfer role within muscle tissue. Although closely related to hemoglobin, myoglobin molecules consist of a single polypeptide chain and contain only one heme group. They therefore lack the ability to bind cooperatively to oxygen or carbon monoxide and have a dissociation curve as represented in Appendix D.²⁸ At low saturation levels, myoglobin binds much more readily with oxygen or carbon monoxide than does hemoglobin.²³ As a result, a person with a carboxyhemoglobin concentration of five to ten percent often has carboxymyoglobin levels about three times higher.⁴ These figures suggest that as much as 30 percent of cardiac myoglobin may typically remain saturated with carbon monoxide in heavy cigarette smokers, whose carboxyhemoglobin levels tend to be about ten percent.²⁷ Although the medical

ramifications of carbon monoxide will be discussed in the next section of this paper, it is quite easy to imagine the impact of such findings. Further, since carboxymyoglobin dissociates slower than carboxyhemoglobin, decreased oxygen reserve in the myocardium of acute poisoning victims is both more severe and more prolonged than simple measurements of carbon monoxide levels in the blood might indicate.⁴ Another result of myoglobin's slow release of carbon monoxide back into the blood is its ability to re-elevate carboxyhemoglobin levels for hours after "normal" levels have been initially re-established in a poisoning victim.⁴ It is therefore important that this be considered prior to discharge.

The accumulation of carbon monoxide in the blood and other tissues has been extensively studied and documented. Needless to say, the greater the percent saturation of carboxyhemoglobin, the more severe the effects of carbon monoxide poisoning. This level is dependent on a number of factors, primarily the concentration of ambient carbon monoxide, the minute ventilation, cardiac output, and the length of exposure.³⁰ In general, carboxyhemoglobin will form relatively quickly at low carbon monoxide saturation levels. Roughly half of all inspired carbon monoxide is immediately retained in the blood at carboxyhemoglobin saturation less than six percent.²⁵ As saturation increases, the rate slows due to decreasing availability of binding sites and the rising back pressure of carbon monoxide already in the blood, until equilibrium is reached (Appendix E). Higher concentrations of inspired carbon monoxide speed carboxyhemoglobin saturation, as do physical exertion, restricted oxygen supply, higher elevations, and numerous other factors. A carbon monoxide concentration of 50 parts per million (ppm), for example, will eventually lead to an equilibrium carboxyhemoglobin level of about seven percent, while 100 ppm leads to 14 percent saturation and 500 ppm results in about 54 percent.³⁰ It should be pointed out, however, that equilibrium is reached only after several hours of relatively constant exposure. Since ambient carbon monoxide concentrations tend to change rapidly in most subacute situations, and since respiration and circulation vary (or halt entirely!) in acute poisonings, equilibrium values are rarely achieved under real conditions.³² None-the-less, they are useful in making comparisons and predictions. Numerous studies, for example, have shown that carbon monoxide levels on many daytime city streets tend to remain between 10 to 15 ppm,

but isolated peaks of 100 ppm or more over short periods are not uncommon. In comparison, the extremely high carbon monoxide concentration of pure cigarette smoke (about three percent by volume, or 30,000 ppm) produces a commonly inhaled mixture of smoke and air that contains between 400 and 500 ppm.³³

An interesting study done at Canada's Defence and Civil Institute of Environmental Medicine in 1987 showed that carbon monoxide uptake in resting humans can be estimated fairly accurately by knowing only the total dose received and the time period over which the exposure occurred. Subjects given a specified number of five-minute doses of 1,500 ppm carbon monoxide experienced the same final carboxyhemoglobin levels as when they received an equal number of one-minute doses at 7,500 ppm over an equal time period.³⁴ Results such as these allow clinicians and medical researchers to more accurately determine what a patient's "initial" (post-exposure) carboxyhemoglobin load must have been by estimating only the total dose received during the exposure time. This eliminates the need to recreate the exposure sequence in more detail or worry about the changes in exposure concentration that likely occurred during the incident.

After an exposure to carbon monoxide has occurred, the gas is slowly released from the blood and other tissues and exhaled. A great deal of research has been done in this area, and has shown that the removal of carbon monoxide follows fairly common decay principles. Breathing normal fresh air, carbon monoxide has a half-life in the blood of about 250 minutes (that is, half the carbon monoxide remaining in the bloodstream will be removed approximately every four hours).⁵ Of even more clinical importance, however, is that this rate of carbon monoxide clearance can be accelerated by administering 100 percent oxygen to poisoning victims, and can be further sped up using hyperbaric chambers to provide 100 percent oxygen at greater pressures (typically about two and a half atmospheres).⁹ This dramatically decreases the mean carbon monoxide half-life to about 50 minutes and 22 minutes respectively.⁵ Appendix F compares these three elimination curves, based on a standard decay function of:

$$\ln \{ [\text{COHb}]_i / [\text{COHb}]_{i+t} \} = kt$$

where \ln is the natural logarithm function, t is elapsed time, k is the decay constant of the particular curve, and $[\text{COHb}]_i$ and $[\text{COHb}]_{i+t}$ are the initial and final carboxyhemoglobin

saturation levels. By working with this basic relationship, it is interesting to note (and critically important to the poisoning victim) that while hyperbaric treatment can remove half of the carbon monoxide from the blood in just 22 minutes, about 94 percent of the original level will still be circulating within the patient if he or she only breaths fresh air for the same time period. The increased oxygen partial pressures delivered by the 100 percent oxygen regimens (both normobaric and hyperbaric) force significant amounts of oxygen to dissolve in the blood plasma. In fact, during hyperbaric treatment enough oxygen is transported by the plasma alone to meet total body oxygen demand, independent of hemoglobin availability. Thus, in addition to hastening CO removal, hyperbaric oxygen can immediately reverse hypoxia.³⁰ Given the desperate need to get oxygen to the heart, central nervous system, and other tissues, these dramatic effects of hyperbaric treatment led to its widespread acceptance in the treatment of severe carbon monoxide poisoning.

Medical Issues

With the above discussion as background, we will now turn to some of the broader effects carbon monoxide has on the body. As previously mentioned, the organs most sensitive to decreased oxygen availability are the heart and central nervous system. Not surprisingly, patients with cardiovascular disease are particularly vulnerable to even "low to moderate" carbon monoxide exposures. In a follow-up study of 63 CO victims, Smith and Brandon noted that a 37 year old man developed a myocardial infarct a few hours after an accidental poisoning, and experienced angina and intermittent congestive heart failure thereafter.³⁵ Another man in the study, a 46 year old, suffered angina on physical exertion following his failed carbon monoxide suicide attempt. Although neither man had problems before their carbon monoxide poisoning, both had family histories of ischemic heart disease.

It was traditionally believed that exposure leading to carboxyhemoglobin levels below 20 percent had negligible effects on the body, but numerous researchers have reshaped some of these ideas. A study in the mid-1970's showed that 40 percent of patients with pre-existing heart disease experienced electrocardiographic abnormalities and decreased angina-limited exercise tolerance when exposed to the Los Angeles freeway for 90 minutes.³⁰

Carboxyhemoglobin levels in all of these individuals were below five percent. In a series of studies, Dr. Poul Astrup, a Clinical Chemistry Professor in Denmark, found that moderate carbon monoxide levels in rabbits (less than 20 percent carboxyhemoglobin) dramatically increased cholesterol accumulation in the arteries.²³ He also showed that these moderate levels cause vascular lesions, possibly by causing intercellular gaps in the arterial walls to widen.²³ Moderate levels of carbon monoxide have also been known to increase the risk of blood clots by decreasing intravascular fluid, altering the fibrinolytic pathway, increasing platelet stickiness, and promoting other cardiovascular changes.¹¹

Studies on carbon monoxide's effects on the central nervous system at low levels have also been noteworthy over the years. Tests on human subjects indicated decreased visual acuity (based on impaired discrimination of small changes in light intensity) at two and four percent carboxyhemoglobin.^{23,36,37} In other performance tests, such as the ability to estimate time intervals or the duration of auditory signals without a clock, individuals have been reported to show impaired abilities at levels around five percent.²³ Although such results may seem insignificant, carboxyhemoglobin levels two to three times higher than these amounts are not uncommon among equipment operators, industrial workers, and automobile drivers, especially in those who are moderate to heavy cigarette smokers. In a lecture I attended in 1988 at the Armed Forces Institute of Pathology, Group Captain A.J.C. Balfour of the Royal Air Force described a fatal nighttime air crash. Although the pilot maintained normal communication with ground personnel, the aircraft gradually veered off course and crashed into the side of a mountain. The mishap investigation determined that equipment malfunction caused a carbon monoxide leak into the crew compartment. At autopsy, the pilot's carboxyhemoglobin level was 20 percent, a level "commonly" associated with headache and the onset of coordination problems.³⁸ Every individual, of course, can experience different symptoms at different levels, but in this particular case a somewhat moderate exposure to carbon monoxide proved fatal for the pilot and his crew.

At higher levels of carboxyhemoglobin saturation, more severe conditions develop in the cardiovascular and central nervous systems, as well as in other areas of the body. Carbon monoxide poisoning can cause cerebral hemorrhage, demyelination of cerebral tissues, and

necrosis of the heart, skeletal muscles, kidney tubules, and brain tissue.^{8,23,30,31,39} Hearing loss may result from hypoxia to the cochlear nerve and brainstem nuclei, while other, less common consequences of carbon monoxide poisoning include acute renal failure, aspiration pneumonia, and peripheral neuropathies.¹¹ Obviously, carbon monoxide victims can suffer a multitude of chronic or acute problems short of asphyxial death. Quite often, those who appear to recover from a near-fatal poisoning develop a wide range of neurological problems weeks or even months after the episode.^{23,40,41,42} Among the more common are alterations in personality and cognitive abilities. In Rome, Italy, for example, a 47 year old fireman suffered an acute CO poisoning in October 1998 while fighting a fire.⁴² He regained consciousness after two days of intensive care, and remained agitated and confused for several days more. Although his neuromuscular exam was nearly back to his pre-exposure condition within several months, he had significant impairment of short-term memory. He had difficulty learning new routes around the city, and required lists in order to accomplish the simplest tasks. He also showed some difficulty orienting himself to well-known places. His wife reported that his personality had become quite apathetic, and that he no longer enjoyed many of his former activities.⁴² In their study of acute poisoning survivors, Smith and Brandon noted that a full third of them experienced personality deterioration after poisoning, while 43 percent reported some degree of memory impairment.³⁵ One of their subjects, a normally developing six year old who was accidentally exposed to carbon monoxide, experienced severe intellectual deterioration and developed a speech defect after the incident.³⁵ Another case was a 33 year old healthy, emotionally stable coal miner who was exposed to carbon monoxide in a mine explosion. He was comatose when found, and was subsequently delirious, aggressive, and irritable for several hours. After receiving oxygen therapy at a nearby hospital, he was released the same day. However, following his carbon monoxide poisoning he became forgetful, began having difficulty coping at work, and was occasionally violent toward his wife. He also became more impulsive, anxious, and paranoid. A few years after the incident was arrested for assaulting five young girls.³⁵ The underlying disease process is suspected to involve progressive, diffuse demyelination of cerebral white matter. Once a certain threshold of demyelination is reached, either through

acute, repetitive, or chronic exposure, an irreversible state is reached and these neurological sequelae become apparent.⁴³ However, the entire pathological mechanism is not yet fully understood, and reliable predictions on an individual basis are difficult at best.

Other functions are affected in addition to the cardiovascular and nervous systems. One key area involves carbon monoxide's effects on the lungs and the potential for long-term respiratory complications. An early study on mice showed that carbon monoxide levels of 50 and 90 ppm began producing progressive changes in their nonciliated bronchiolar cells and alveolar cells in as little as 17 hours.⁴⁴ Ambient carbon monoxide concentrations in this range can be common in many urban areas, and, over time, lead to carboxyhemoglobin levels of about seven to fourteen percent in humans (Appendix E). Necrosis of sweat glands has also been reported in carbon monoxide victims, and may severely impact the body's temperature regulation.³⁹ In one particular study, all of the patients who died from carbon monoxide poisoning were found to have body temperatures of 102 degrees Fahrenheit or higher.³⁹ Work in this area has led to the use of hypothermia in the treatment of some poisoning cases, which reportedly can prevent or reverse some of the adverse effects on the central nervous system.³⁹

A final area that deserves attention is carbon monoxide exposure during pregnancy, and its effects on fetal and, later, neonatal development. Easily crossing the placenta into the fetal circulation, CO has been shown to decrease placental blood flow, thereby further hampering oxygenation of fetal tissue. Combined with its well-documented ability to damage neural structures, and considering the vulnerabilities of the developing fetal brain, there is little wonder that even moderate levels of CO can have devastating effects, especially during the first trimester. It has been shown that mothers who smoke during pregnancy have more premature births, lower birth-weight babies, and more often have infants with delayed crying or other respiratory irregularities.^{23,45} Their babies are also more likely to develop respiratory illnesses during the first year of life.²³ Various researchers studying rabbits, monkeys, and other animal models have observed lower fertility rates, smaller litter size, and alarmingly higher rates of spontaneous abortion, fetal death, physical malformations, and severe neurological impairment at various maternal CO exposure levels and frequencies.^{23,45}

Epidemiological studies indicate human results are fairly consistent with these experimental findings. Fetal and neonatal deaths, limb malformations, various functional impairments, and delayed or stunted cognitive development have been well-documented following chronic maternal exposure or significant acute intoxication^{45,46,47,48} Further, the vasodilatory effects of carbon monoxide has been shown capable of causing postpartum hemorrhage, and perhaps other serious complications in the mother.⁴⁹ Still other researchers have proposed some possible connections between carbon monoxide in the home and sudden infant death syndrome (SIDS).⁵⁰ These proposals are based on findings that some SIDS cases may be due to a breakdown in the infant's ability to properly integrate life-sustaining regulatory systems, and that exposure to low levels of carbon monoxide could be enough to shut down respiratory control. They note that mothers who smoke cigarettes during pregnancy and after delivery have been reported in 41 to 71 percent of SIDS cases.⁵⁰ Further, more SIDS cases occur between October and March, and in lower socioeconomic homes, which also suggests carbon monoxide or other environmental factors are directly involved. Although a great deal of additional work is needed in this area, it has been noted that some of the pathologic abnormalities of both SIDS and carbon monoxide poisoning are similar to, and consistent with, chronic hypoxia.⁵⁰

BEXAR COUNTY DATA

In order to appreciate the public health dimension of any particular issue a "public," or population must be defined. This helps an interested reader, researcher, or policy maker to consider the specified health risk (be it a microbe, a disease vector, a safety hazard, or an environmental toxin) in a relevant context. For this particular review of the hazards of carbon monoxide, the chosen setting was the CO experiences in Bexar County, Texas over the past several decades. Bexar County, which includes the city of San Antonio, has a current population of about 1.4 million, as reported in the 2000 census.⁵¹ The goal was to present a descriptive epidemiological study of the county's nearly 600 CO deaths that occurred between 1936 and 1995. A more detailed demographic evaluation of the most

recent ten-year period (1986 to 1995) was accomplished to identify any higher risk groups for possible "targeted" public health education efforts.

Data

Data for this study were obtained from an existing, anonymous data set maintained by the San Antonio campus of the University of Texas Health Science Center at Houston's School of Public Health. In the latter part of the 1980's, Bexar County death records from 1935 through 1985 were compiled, coded, and edited with the support of a grant by the National Institute of Child Health and Human Development, and have been used in several local studies since that time. Following the initial compilation, records for 1986 through 1995 have been provided periodically to the School of Public Health by the Texas Department of Health, and the database has been updated accordingly.

Each death record in the database (n=369,900) provided the year of death, year of birth, and age at death for each of the deceased, along with a random numeric identification code assigned for tracking purposes. Also shown were the underlying cause of death and up to 14 associated causes of death (by ICD-9 codes), plus basic demographic data such as gender, race, marital status, and general residence area (listed as "San Antonio," "other urban county areas," "rural Bexar County," "other Texas county," etc.). Fields were also present for listing broad occupational areas and for identifying deaths from job-related injuries, but unfortunately the entries in these fields were sporadic and quite incomplete. From this database a total of 588 cases were identified and extracted which involved carbon monoxide poisoning. Although carbon monoxide was not the underlying (primary) cause of death in every instance, a "case" was defined as a death in which either the underlying or one of the associated causes of death included a reference to CO toxicity. A listing of the ICD-9 codes (and their corresponding text descriptions) used during the database search, as well as those incidentally encountered in the extracted cases, is shown in Appendix G. The 588 cases and the codes for the table listings are provided for reference in Appendices H and I.

Historical population data for Bexar County were obtained from U.S. Census Bureau statistics.^{51,52} Population data of interest included total county population figures for each of

the decennial censuses from 1900-2000 and county sub-population estimates by age, sex, race, and Hispanic ethnicity for 1990. These data are shown in Appendices J and K.

Methods

Based on the ICD-9 codings, the manner of death was determined for each of the 588 cases and added to the data as the new variable **mod**. "Manner of death" classifies a given death as either natural, accidental, suicide, homicide, or undetermined. Obviously, the nature of carbon monoxide poisoning essentially excludes the possibility of any cases being truly "natural" deaths. Homicides involving use of CO to poison another person occur from time to time, but none were found among the Bexar County data. Therefore, all 588 cases were classified as either suicidal ($n_s=321$), accidental ($n_a=258$), or undetermined ($n_u=9$). Because of the uncertainty surrounding the nine undetermined deaths, they have been listed to allow a more detailed review in Appendix L. These deaths, as well as several other cases involving somewhat unusual or "interesting" circumstances, are discussed in more detail below.

To streamline the analysis the study period was divided into 12 separate five-year groups: "1936-1940" through "1991-1996," with the death data grouped accordingly. The year 1935 was omitted so that 12 equal periods could be evaluated and compared. It should be noted that one CO-related death, the suicide of a 57 year old non-Hispanic white female, occurred that year. (This would have represented a 589th case.)

Using the census figures for Bexar County (Appendix J), population estimates for the central year of each five-year study period were determined by linear interpolation between the corresponding decennial censuses. For example, the population for 1938 (central year of the 1936-1940 period) was estimated as: $P_{1938} = P_{1930} + (0.8)(P_{1940} - P_{1930})$. Similar calculations were done for each of the 12 periods studied. This allowed standardization of crude five-year death rates to five-year deaths per million residents.

Following calculation of the overall death rates by manner of death for the 60-year study period, a more detailed review of the final ten years (1986-1995) was done in order to highlight any recent trends or higher risk groups. Demographic factors of specific interest included age, sex, race, Hispanic ethnicity, occupation, and occupational-related CO

exposure. An attempt was also made to detect and evaluate instances of multiple-victim poisonings.

Results

The carbon monoxide deaths experienced in Bexar County during each year from 1936 through 1995 are listed in Table 1 below, and are shown graphically in Figure 1 on the next page. The annual breakdown by manner of death for each year is also shown.

The raw death figures were then grouped into 12 five-year periods (1936-40 through 1991-95) to aid evaluation and comparison. These grouped results are shown on Table 2 on page 16, and in Figure 2 on page 17.

As described previously, the crude five-year death rates from Table 2 and Figure 2 were then standardized to account for population growth in Bexar County during the 60-year period being reviewed. Overall carbon monoxide death rates, along with the rates for each manner of death, were calculated per million county residents for the 12 five-year periods. The results, which allow clearer comparisons of rates and trends than do the raw data, are

Year	Sui.	Acc.	Und.	Total	Year	Sui.	Acc.	Und.	Total	Year	Sui.	Acc.	Und.	Total
1936	4	1	0	5	1956	3	3	0	6	1976	7	8	2	17
1937	1	0	0	1	1957	2	13	0	15	1977	6	4	0	10
1938	1	1	0	2	1958	4	9	0	13	1978	6	5	0	11
1939	0	3	0	3	1959	4	13	0	17	1979	3	7	0	10
1940	0	1	0	1	1960	5	9	0	14	1980	4	9	1	14
1941	2	0	0	2	1961	7	9	0	16	1981	3	6	0	9
1942	0	1	0	1	1962	11	9	0	20	1982	7	3	0	10
1943	1	0	0	1	1963	12	7	0	19	1983	7	4	0	11
1944	2	2	0	4	1964	6	2	0	8	1984	8	3	0	11
1945	2	3	0	5	1965	7	5	0	12	1985	10	3	0	13
1946	2	2	0	4	1966	21	5	1	27	1986	10	1	0	11
1947	2	0	0	2	1967	8	15	0	23	1987	8	0	0	8
1948	2	2	0	4	1968	4	14	1	19	1988	9	1	0	10
1949	3	1	0	4	1969	6	11	0	17	1989	10	7	0	17
1950	2	1	0	3	1970	4	6	1	11	1990	4	3	0	7
1951	0	1	0	1	1971	9	9	0	18	1991	10	3	0	13
1952	0	0	0	0	1972	5	9	0	14	1992	7	0	1	8
1953	5	0	0	5	1973	11	7	0	18	1993	6	7	1	14
1954	4	0	0	4	1974	4	2	0	6	1994	6	0	0	6
1955	5	0	0	5	1975	9	7	1	17	1995	10	1	0	11
										SUM	321	258	9	588

Table 1. Annual carbon monoxide deaths in Bexar County by manner of death, 1936-1995

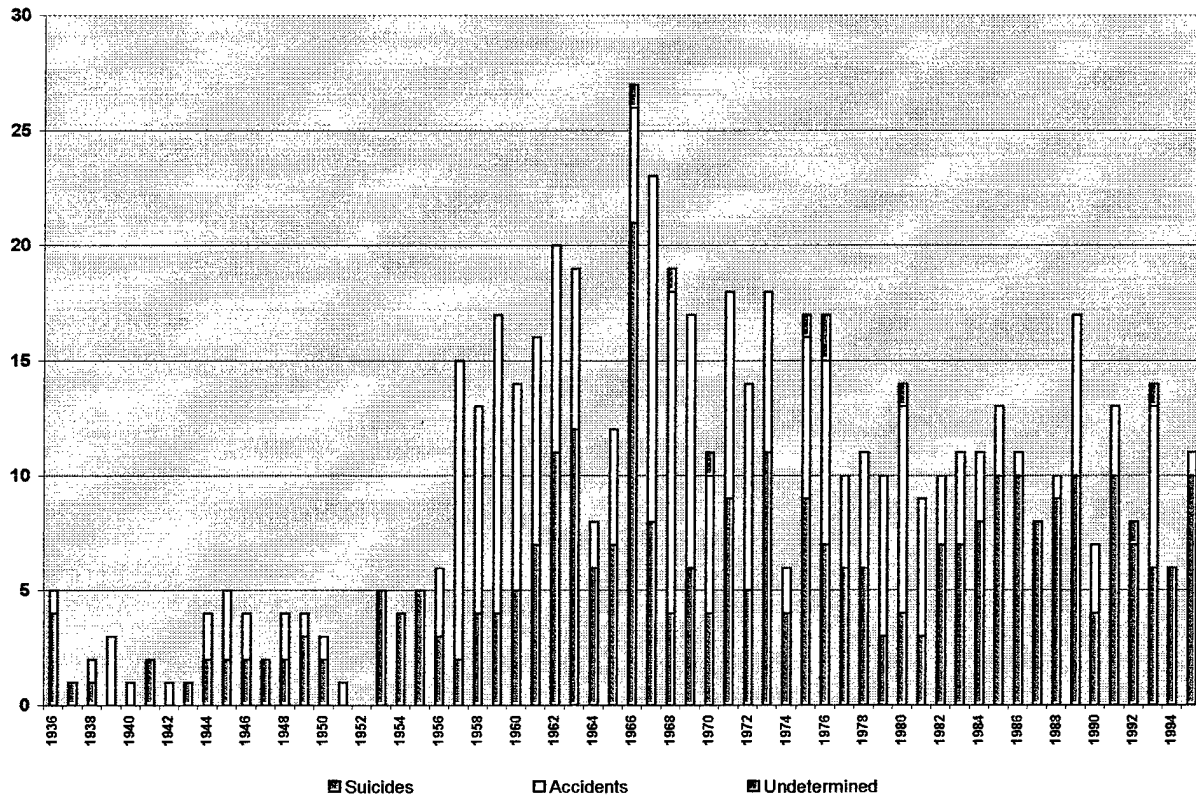


Figure 1. Annual carbon monoxide deaths in Bexar County, by manner of death, 1936-1995

shown in Table 3 and Figure 3 on page 18.

Overall, the carbon monoxide related death rates in Bexar County during the 60 year period 1936 to 1995 were relatively stable at about 30 to 40 deaths per million residents

Years	Suicide	Accidental	Undetermined	TOTAL
1936-1940	6	6	0	12
1941-1945	7	6	0	13
1946-1950	11	6	0	17
1951-1955	14	1	0	15
1956-1960	18	47	0	65
1961-1965	43	32	0	75
1966-1970	43	51	3	97
1971-1975	38	34	1	73
1976-1980	26	33	3	62
1981-1985	35	19	0	54
1986-1990	41	12	0	53
1991-1995	39	11	2	52
Total	321	258	9	588

Table 2. Five-year CO death rates in Bexar County by manner of death, 1936-40 to 1991-95

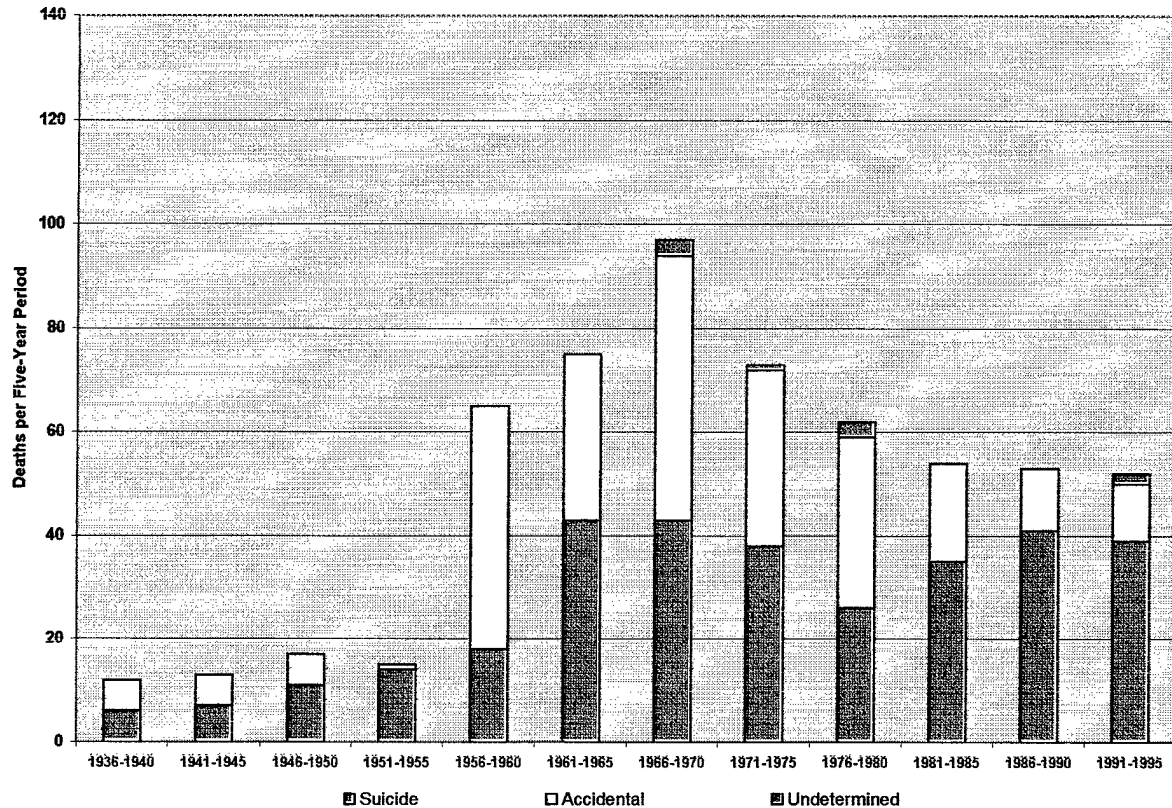


Figure 2. Five-year CO death rates in Bexar County, by manner of death, 1936-40 to 1991-95

during each five year period from 1936-40 through 1951-55. This was followed by a marked increase (3 to 4 times these rates) from 1956-60 through 1966-70. The highest death rate was 121 deaths per million, which occurred during the 1966-1970 period. These high rates were followed by a gradual decline back to about 40 deaths per million per five years by the final period of the study, 1991-95. Much of the increase noted between 1956 and 1970 was due to higher numbers of accidental deaths. Accidental deaths then fell markedly, and have remained quite low since 1980. An increase in rates of carbon monoxide suicides is also apparent during the 1960's and early 1970's. Suicide rates peaked about the same time as accident rates; however, accidental death rates increased and declined more rapidly than suicides. As a result, accidental carbon monoxide death rates have recently fallen below the

Years	Suicide	Accidental	Undetermined	TOTAL
1936-1940	18.23	18.23	0	36.47
1941-1945	18.09	15.51	0	33.60
1946-1950	23.50	12.82	0	36.32
1951-1955	25.16	1.80	0	26.96
1956-1960	27.70	72.33	0	100.03
1961-1965	58.89	43.83	0	102.72
1966-1970	53.63	63.61	3.74	120.98
1971-1975	43.28	38.73	1.14	83.15
1976-1980	27.16	34.48	3.13	64.78
1981-1985	33.40	18.13	0	51.54
1986-1990	35.77	10.47	0	46.24
1991-1995	31.26	8.82	1.60	41.68

Table 3. Five-year CO death rates in Bexar County, by manner of death, per million residents, 1936-40 to 1991-95

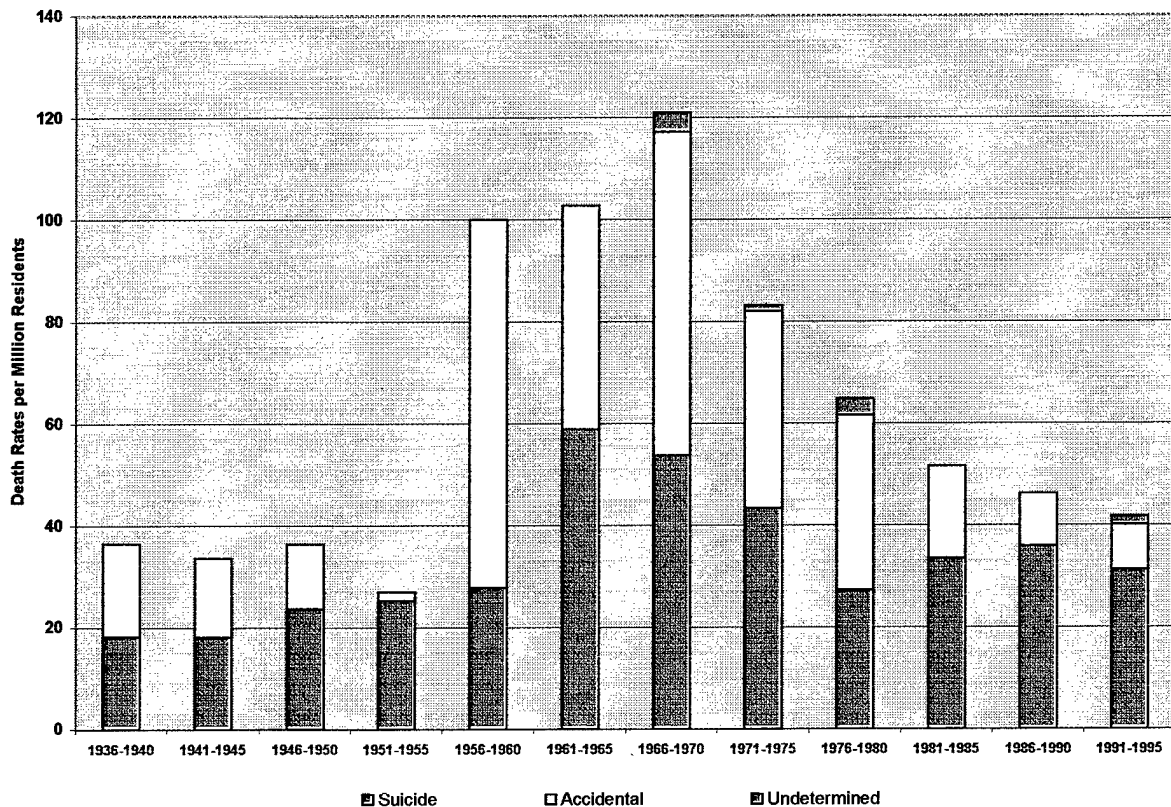


Figure 3. Five-year CO death rates in Bexar County, by manner of death, per million residents, 1936-40 to 1991-95

rates noted for the 1930's and 1940's, while suicides have remained approximately one and a half times as high as their pre-1950 rates.

Closer inspection of the data for 1986-1995 examined the various sub-populations by age, sex, race, and Hispanic ethnicity. It was originally hoped that a comparison could be made between occupational-related accidental deaths and domestic accidents, and that higher risk occupations could be identified. Unfortunately, the data presented some very serious limitations here. Of the 588 total cases there were only six work-related accidental deaths listed. Five others were occupational-related CO suicides, while 419 were coded as not work-related. In 71 cases no information regarding occupational-relatedness had been listed on the death certificates, and one death was coded as "unknown." The greatest obstacle, however, was that 86 cases (all occurring between 1986 and 1995, and accounting for 82 percent of the cases during that period) were coded improperly. As shown in Appendices H and I, they were coded as "2," which was not a defined value for this variable. Attempts to interpret and correct the miscoding with any degree of confidence were unsuccessful.

Other demographic considerations for the 1986-1995 period were more fruitful. Table 4 below, and Figure 4 on page 20, show the gender variation among the 588 carbon monoxide deaths studied. When gender is regarded as a single variable, the data indicate that males die from accidental CO exposure five times more often than females, and commit suicide using carbon monoxide more than two and a half times as often. In order to more fully explore these variations, gender differences were examined in each of the demographic evaluations (age, ethnicity, and race) that followed.

The distribution of CO deaths by 10-year age groups ranging from pre-teen (0-9 years) through age "70 and over" is shown in Table 5 and Figure 5 on page 21. These data indicate a noticeable trend in the distribution of carbon monoxide deaths that increases to a high in the

	Suicide		Accidental		Undetermined		ALL MANNERS	
	Deaths	per Million	Deaths	per Million	Deaths	per Million	Deaths	per Million
All Males	57	98.94	19	32.98	1	1.74	77	133.66
All Females	23	37.61	4	6.54	1	1.64	28	45.79
Total	80	67.36	23	19.37	2	1.68	105	88.41

Table 4. Ten-year CO death rates in Bexar County by sex and manner of death, 1986-1995

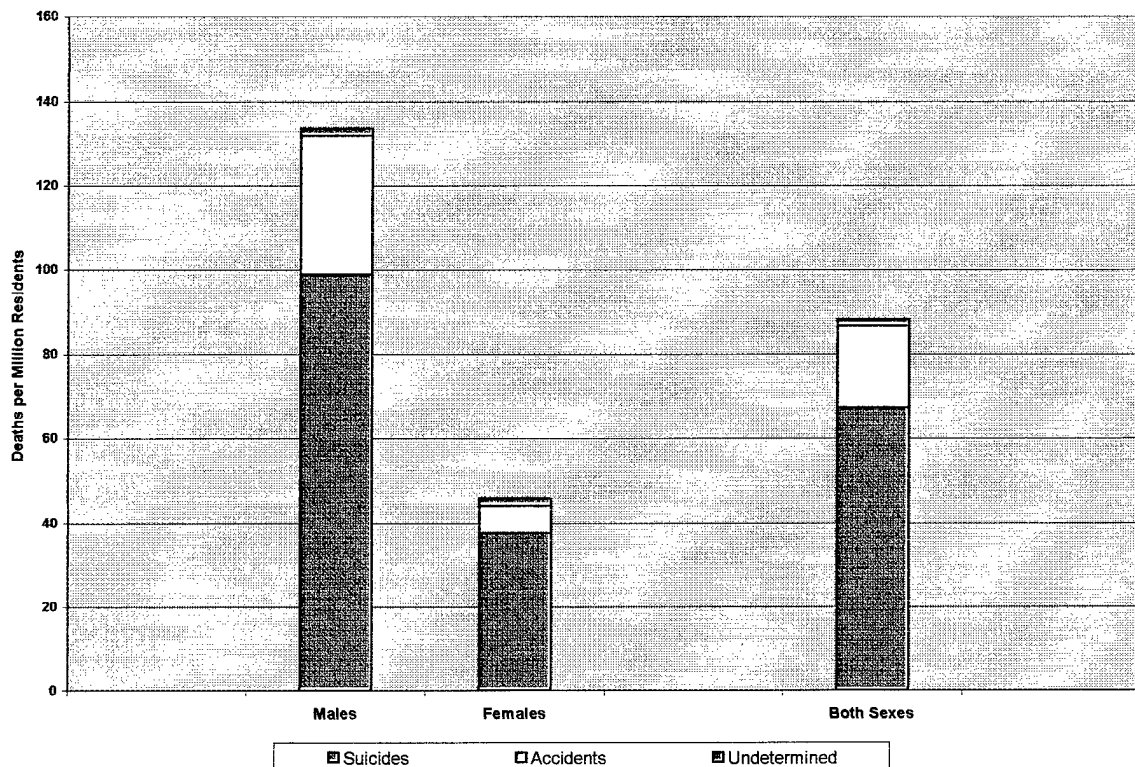


Figure 4. Ten-year CO death rates in Bexar County by sex and manner of death, 1986-1995

fourth decade of life (age 30-39), and then essentially levels for several decades before dropping to about 50 deaths per million among those age 70 and over. Interestingly, this most elderly group experienced approximately equal rates of CO suicides and accidental deaths. Among the very young, teenagers experienced carbon monoxide suicides, but did not succumb to accidental CO poisonings during the 1986-1995 period. Not too surprisingly, there were no CO suicides in pre-teens.

Regarding racial differences, it should first be noted that in Bexar County Hispanic whites and non-Hispanic whites account for 91 percent of the county's total population (49.1 percent are Hispanic whites and 41.8 percent are non-Hispanic whites), with the next largest sub-population being blacks (7.3 percent of the total population).⁵² Therefore, Hispanic ethnicity (rather than race *per se*) has traditionally played a greater demographic role in this

	Suicide		Accidental		Undetermined		ALL MANNERS	
	Deaths	per Million	Deaths	per Million	Deaths	per Million	Deaths	per Million
All age 0-9	0	0.00	1	4.97	0	0.00	1	4.97
Male	0	0.00	0	0.00	0	0.00	0	0.00
Female	0	0.00	1	10.13	0	0.00	1	10.13
All age 10-19	8	42.33	0	0.00	0	0.00	8	42.33
Male	5	51.70	0	0.00	0	0.00	5	51.70
Female	3	32.51	0	0.00	0	0.00	3	32.51
All age 20-29	16	78.53	6	29.45	0	0.00	22	107.98
Male	12	118.26	5	49.28	0	0.00	17	167.54
Female	4	39.11	1	9.78	0	0.00	5	48.89
All age 30-39	24	122.50	3	15.31	1	5.10	28	142.91
Male	20	209.52	2	20.95	1	10.48	23	240.95
Female	4	39.81	1	9.95	0	0.00	5	49.77
All age 40-49	13	91.41	5	35.16	0	0.00	18	126.57
Male	7	102.19	4	58.40	0	0.00	11	160.59
Female	6	81.39	1	13.57	0	0.00	7	94.96
All age 50-59	10	105.81	2	21.16	0	0.00	12	126.97
Male	9	201.09	2	44.69	0	0.00	11	245.77
Female	1	20.10	0	0.00	0	0.00	1	20.10
All age 60-69	7	82.85	3	35.51	1	11.84	11	130.19
Male	3	80.69	3	80.69	0	0.00	6	161.38
Female	4	84.54	0	0.00	1	21.14	5	105.68
All age 70 & over	2	26.20	2	26.20	0	0.00	4	52.41
Male	1	34.06	2	68.13	0	0.00	3	102.19
Female	1	21.29	0	0.00	0	0.00	1	21.29
TOTAL	80	71.99	23	20.70	2	1.80	105	94.49
Male All Ages	57	104.25	19	34.75	1	1.83	77	140.83
Female All Ages	23	40.74	4	7.09	1	1.77	28	49.60

Table 5. Ten-year CO death rates in Bexar County by age, sex, and manner of death, 1986-1995

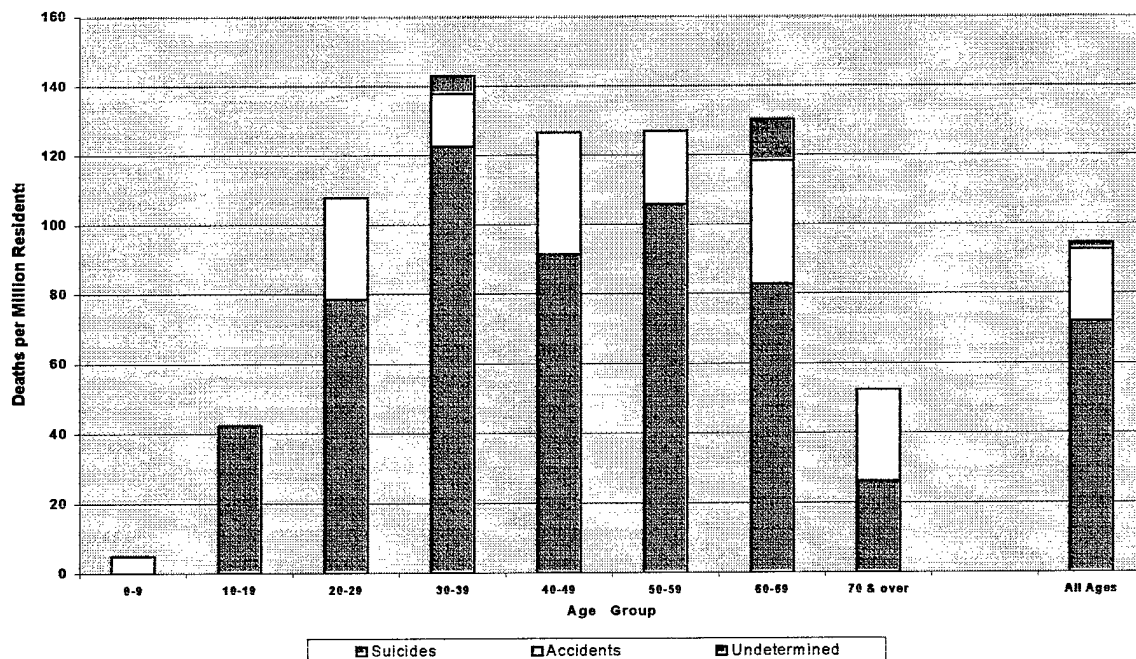


Figure 5. Ten-year CO death rates in Bexar County by age and manner of death, 1986-1995

part of Texas. Furthermore, census data report that 98.5 percent of Bexar County Hispanics are racially white (see Appendix K). Therefore, for the purposes of this study, I considered race and ethnicity using the following four groups: Hispanic whites, non-Hispanic whites, blacks, and "other." Results for race and Hispanic ethnicity are shown on Table 6 below, and Figure 6 on page 23.

As shown, the death rates due to accidental carbon monoxide poisoning are quite similar for Hispanics, non-Hispanics, and blacks (18 to 23 deaths per million during 1986-1995), and rate of 48 deaths per million overall, and 85 deaths per million among "other race" females.

Suicide rates showed some interesting racial and ethnic differences. Carbon monoxide suicides within the Hispanic population were quite lower, for both sexes, than among non-Hispanics whites. In fact, non-Hispanic white men intentionally killed themselves by this method nearly eight times more often than their Hispanic neighbors. The difference among women in these two groups was even more startling: the non-Hispanic white female suicide rate by carbon monoxide was twelve times as high as that for Hispanic female CO suicides. Blacks, as a race, more closely resembled the suicide rates of Hispanics. A closer look, however, revealed there were only two black suicides by CO, and both were males. This resulted in a black male suicide rate twice as high as Hispanic males, but still only a quarter as high as non-Hispanic white males. Suicides among members of "other" races consisted of

	Suicide		Accidental		Undetermined		ALL MANNERS	
	Deaths	per Million	Deaths	per Million	Deaths	per Million	Deaths	per Million
Hispanic Whites	9	15.42	11	18.85	1	1.71	21	35.99
Male	7	24.72	9	31.78	0	0.00	16	56.49
Female	2	6.66	2	6.66	1	3.33	5	16.65
Non-Hisp. Whites	67	134.87	9	18.12	1	2.01	77	155.00
Male	46	190.05	9	37.18	1	4.13	56	231.36
Female	21	82.44	0	0.00	0	0.00	21	82.44
Blacks	2	23.19	2	23.19	0	0.00	4	46.38
Male	2	48.14	1	24.07	0	0.00	3	72.21
Female	0	0.00	1	22.38	0	0.00	1	22.38
All Other Races	2	95.08	1	47.54	0	0.00	3	142.63
Male	2	215.49	0	0.00	0	0.00	2	215.49
Female	0	0.00	1	85.08	0	0.00	1	85.08
Total All Races	80	67.36	23	19.37	2	1.68	105	88.41
Male	57	98.94	19	32.98	1	1.74	77	133.66
Female	23	37.61	4	6.54	1	1.64	28	45.79

Table 6. Ten-year CO death rates in Bexar County by race or Hispanic ethnicity, sex, and manner of death, 1986-1995

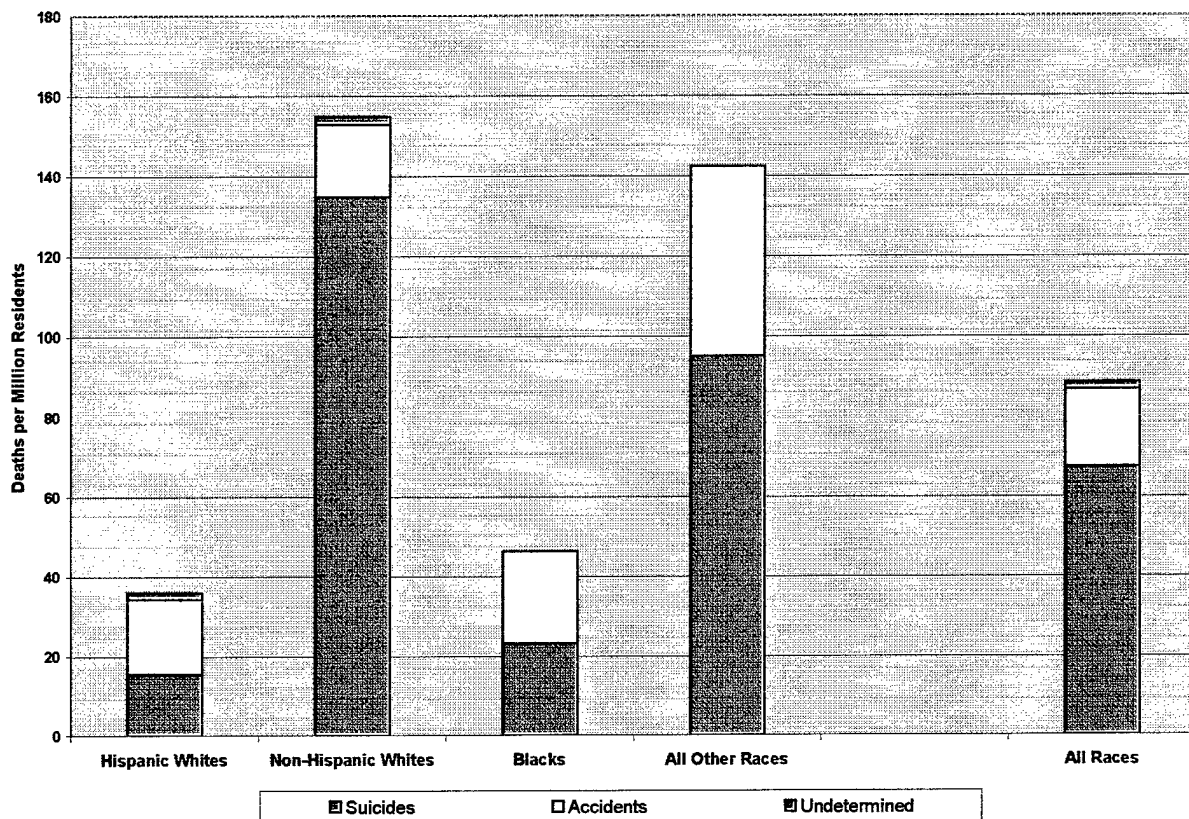


Figure 6. Ten-year CO death rates in Bexar County by race or Hispanic ethnicity and manner of death, 1986-1995

only two, both committed by Asian males. Given the small percentage of “other” racial groups in the county (less than 2 percent), these two suicides produced the rates that are shown in Table 6 and Figure 6.

Discussion

Important issues in any fatal event are the determinations of cause and manner of death. *Cause of death* deals with the pathological processes that brought about the person’s demise, while *manner of death* is a medicolegal determination as to the circumstances surrounding the processes that led to the death. This study specifically selected the 588 deaths that occurred in Bexar County, Texas between 1936 and 1995 that involved carbon monoxide

toxicity as either the underlying or an associated cause of death. These cases were then divided according to manner of death (natural, accidental, suicide, homicide, or undetermined). As previously mentioned, deaths due to carbon monoxide poisoning are, by definition, not "natural." However, relatively low CO levels can and do become lethal for individuals with compromised cardiac or respiratory functions, as well as other acute or chronic illnesses. The 588 cases reviewed in this study revealed several deaths involving such a combination of acute carbon monoxide exposure and pre-existing medical conditions. A 76 year old woman died in 1959 of an acute myocardial infarct and CO toxicity, and a 66 year old man with unspecified cardiovascular disease died of cardiac complications after a carbon monoxide exposure in 1990. In 1975, a 25 year old man with pneumonia died when the illness was aggravated by carbon monoxide. In all of these cases, since the CO exposures were unintentional the deaths were appropriately ruled accidental. Even if the carbon monoxide levels may have been non-lethal in healthier victims, the natural disease processes alone would not have caused the patients' deaths at that particular time had it not been for the added stress of the carbon monoxide. A more complicated case, however, involved a 23 year old man whose primary cause of death was thyrotoxicosis. He also had cardiac dysrhythmias (and ultimately cardiac arrest), a carbon monoxide exposure, and an unspecified trauma which brought about an intracranial hemorrhage and a liver injury. A variety of possible scenarios could be imagined leading to this man's demise in 1970, but, without further information (and primarily because the cause of the carbon monoxide exposure was listed as "undetermined whether intentional or accidental"), the manner of death was classified as undetermined. Overall, nine of the 588 carbon monoxide associated deaths in this study were listed as undetermined. Each was reviewed in as much detail as available, and summarized in Appendix L.

Before discussing suicidal and accidental CO fatalities, a few words about homicides should be mentioned. As noted earlier, homicidal carbon monoxide poisonings are relatively uncommon but certainly do occur from time to time. Although none were reported or identified in Bexar County from 1936-1995, a news item from the west coast appeared while I was compiling information for this thesis.¹⁸ According to the report, a California father was

charged with killing five of his six children (ages 2 to 10) in an attempted murder/suicide by intentionally lighting a charcoal grill in the living room. Only the man and a nine-year-old daughter survived, and both remained hospitalized. The investigation ruled out any possible heating or cooking efforts; homicidal intent was evident, and motive appeared to be related to marital discord.

Regarding accidental CO poisonings, it was not surprising that the vast majority of the unintentional deaths involved fires in private dwellings or carbon monoxide ingestion from other conflagrations. Several of the rarer accidents included six separate instances of accidental ignition of clothing, and two cases of bed linen fires. In 1967, a 59 year old woman died of alcohol dependence syndrome, with an associated carbon monoxide poisoning. As mentioned earlier, data limitations hindered a comparison of domestic versus occupational carbon monoxide deaths, in that needed data was unavailable for 157 (27%) of the cases. Of the six known work-related CO fatalities from 1936-1995, all were men between the ages of 31 and 61, and no two cases occurred in the same year. Occupations of the victims included two transportation/equipment operators (both died from accidental exposure to motor vehicle exhaust), two general service workers (no information other than "carbon monoxide intoxication" was available), and two who worked as management or administrative personnel (one died from motor vehicle exhaust, while the other was a passenger on an aircraft when the CO exposure occurred). Overall, accidental deaths accounted for 258 of the 588 carbon monoxide fatalities (44 percent) reported in Bexar County from 1936 through 1995.

During this same 60-year period Bexar County experienced 321 suicides involving carbon monoxide (55 percent of the total CO deaths). Many involved the use of automobile exhaust or combustion gases from domestic fuels, and at least five occurred at the individual's place of employment or involved work vehicles. In five other cases, rather determined individuals employed multiple methods to ensure their deaths: three were coded as suicidal hangings with accompanying carbon monoxide, and two were listed as primarily suffocation deaths with CO as an associated cause. A lengthy discussion regarding suicidal methods of choice is beyond the scope of this study; however, it should be pointed out that

the rates of carbon monoxide suicides among the various demographic groups (and among individuals in any group or geographic area) should not be expected to be indicative of suicide rates in general. On the other hand, although carbon monoxide is not typically among the top suicidal methods of choice in this country, it does, unfortunately, offer a painless, inexpensive, easily available, relatively discreet, and extremely lethal option to many who are seeking self-destruction.

Regarding the trends in the carbon monoxide deaths noted for Bexar County from 1936-1995, several possible explanations seem reasonable, although additional data or broader studies would be needed to confirm them. As noted earlier, there was a sharp rise in accidental poisonings in the late 1950's, followed by a gradual decrease after 1970 to levels even lower than those of the earliest period studied (1936-1940). This seems to coincide with trends in urbanization, industrialization, and a general increased reliance on mechanized equipment (both at home and at work) following World War II. Later, increasing hazard awareness, improved consumer and product safety, stricter air quality standards, and lower emission levels likely brought about and sustained the lower accident rates. As for suicide trends, once again the motives for selecting a particular method of suicide can be quite variable. Certain underlying factors as personal stress levels, increasing social complexity or disorder, growing population density, civil unrest, wars, and political turmoil could be expected to effect the overall incidence of suicide in a given population. And, as already noted, different racial or ethnic groups have noticeably higher or lower rates. Further, some of the things that effect the incidence of a *specific* suicide method include availability of items needed to enact the attempt, the modeling or "copying" of recent or familiar suicides, and the individual's level of impulsivity as well as other mental status considerations. Any of these, and no doubt several others, likely had a role in influencing the carbon monoxide suicide trends observed in Bexar County. Also, use of direct adjusted rates may have identified additional differences in death rates due to gradual changes in the age or other demographic composition of the study population. Future work in this area may reveal additional, interesting findings.

Another area of particular interest was the occurrence of any multiple or "mass" carbon monoxide poisonings. Occasionally, two or more deaths with similar underlying causes and demographic characteristics appear sequentially in the database (see Appendix H), but unfortunately there were no specific links in the existing data set to fully identify such events. An extensive study of the original death certificates was outside the proposed scope of this investigation, but would have been able to detect concurrent deaths. However, in addition to the recent California murder case mentioned earlier, there was a multiple carbon monoxide death case in San Antonio in early 2002.^{16,17} One of a pair of friends left his car running in the garage, reportedly to charge the battery, and apparently forgot to turn it off. Both men subsequently died during the night, and were found by a female friend the following morning. Fortunately, many other cases of multiple intoxications have happier endings, with someone recognizing the problem in time, and all (or most) of the victims survive. I was told informally of one such family, treated in early 2002 at Wilford Hall Medical Center, one of San Antonio's three level one trauma centers. The family members included several children and adults, and each victim in that case was discharged with a good prognosis after appropriate treatment, testing, and observation.

Given the background information and the results obtained through this study, questions regarding ways to improve public awareness and education seem to naturally follow. Utility companies, for example, occupy an important position in this regard. As suppliers of energy and fuels, particularly fuels for domestic use, utility companies are in a position to not only educate their customers about relevant hazards, they can take an active role in public health and safety. I was therefore pleased to see a carbon monoxide warning on my December 2001 bill from City Public Service (CPS). An announcement, safety warning, or energy conservation tip is generally printed on each monthly statement. The December warning read:

***** CARBON MONOXIDE (CO) POISONING *****

A DIRTY OR FAULTY HEATING SYSTEM CAN PRODUCE CARBON MONOXIDE. THE COLORLESS, TASTELESS AND ODORLESS GAS PROVOKES FLU-LIKE SYMPTOMS. EXPOSURE TO CARBON MONOXIDE (CO) FOR EXTENDED PERIODS CAN LEAD TO UNCONSCIOUSNESS, BRAIN DAMAGE OR DEATH. PLEASE HAVE YOUR HEATING EQUIPMENT CHECKED NOW BY A LICENSED CONTRACTOR.

The warning hit the most important points, including symptoms to watch for, how serious CO poisoning can be, and how easily the risk can be minimized. I called CPS to find out how one might report a "suspected carbon monoxide leak," and learned that CPS does not test, repair, or respond in any other way to problems inside an individual home. Other than promising to send a list of potential servicing contractors, they offer customers nothing in the way of emergency referral or other suggested actions. Interestingly, six weeks elapsed before the list was mailed, and a few days more before I received it. Fortunately, I had no suspected leak. I also felt fortunate that I could have found a contractor on my own if needed, and that I knew a little more about carbon monoxide than the average CPS customer. Overall, CPS gets credit for sending out a nice hazard warning, but they might consider being a bit more helpful if a customer actually thinks he has a problem. I found myself wondering how responsive utility companies are in other (colder) parts of the country.

Another area that lends itself to public education and hazard awareness is consumer product information and warnings. A large number of common household items generate carbon monoxide, even when functioning properly (charcoal grills, gasoline generators, heating units, lawn mowers, and so forth). Indeed, charcoal briquettes seem to have a particularly notorious place in the accidental CO literature.^{1,3}

The stated (or perhaps the "traditional") intent of instruction booklets, operators' manuals, product information bulletins, and similar publications is to instruct on the safe and proper use of the item. They raise the user's awareness of inherent hazards, and provide specific, appropriate warnings. In other words, they are intended to protect public health. Admittedly, the truest reason for most of what is written in these booklets and on warning labels these days is likely aimed at "tertiary prevention" ...of lawsuits. Never the less, an informal survey of several product information sheets revealed, for the most part, adequate warnings about the production of CO and prevention of CO poisoning. Charcoal is not to be lit or used indoors, or even outdoors in poorly ventilated areas; gasoline-powered chainsaws, leaf-blowers, snow-throwers, air compressors, and generators universally had carbon monoxide warnings in addition to the required cautions about keeping any moving blades away from your face, pets, and small children. A few warnings even made reference to how

quickly CO can build to dangerous levels in confined spaces, and all of them warned users not to leave the products operating when unattended. Warnings seem, therefore, to be “available” and of appropriate content. The key issues remaining, of course, are compliance and “operator error.”

Public awareness and behavior is also influenced by the media. Aside from barber shop banter and impromptu group-therapy sessions during happy hour, the average citizen tends to rely on television news broadcasts, local papers, the Internet, and TV commercials for much of his or her information. Once again, an informal survey of some of these media was done to detect the topic of carbon monoxide, and any warnings as to its public health threat. Not surprisingly, news reports, both televised and in print, tend to only occur after a local incident. As an addendum to a brief report on the two San Antonio men killed this past January, one local news station aired a two-minute “special report” on the dangers of carbon monoxide exposure. While it most likely helped raise viewers’ awareness, some of the statements made could have been a little more accurate had the reporter spent more time learning the facts rather than focusing on the dramatics. A particularly interesting quote was:

Eventually what happens, the experts tell me, is that the carbon monoxide prevents the oxygen from getting into the lungs. So they describe it this way: that a person essentially suffocates from the inside out!¹⁶

Highway billboards may offer another way to keep the public thinking of hazards like carbon monoxide. Ads concerning diabetes, symptoms of heart attack and stroke, the dangers of smoking, signs of depression, cosmetic surgery options, pregnancy counseling, breast reduction, vasectomy reversal, and a multitude of other vital health concerns are already quite visible along Bexar County highways. Why not carbon monoxide?

CONCLUSION

Claiming the lives of three to four thousand Americans each year, carbon monoxide is the leading cause of poisoning deaths in this country. Interestingly, it does not seem to get

the same attention as many other hazardous substances, perhaps *because* of its perceived familiarity. Never the less, it remains an ever-present danger; and a “silent killer.”

Although this particular study focused on fatal carbon monoxide poisonings, it is important to remember that there are far greater numbers of victims than those most unfortunate ones. The sub-lethal effects and long-term consequences of CO intoxication are many, varied, and often only slightly less damaging than death.

The recent carbon monoxide deaths in Bexar County, Texas, seem fairly consistent with national trends, considering its generally warmer climate and its demographic make-up. This is not to imply, however, that continued vigilance and public awareness education should not continue (or begin!). Such awareness may be the only key to detecting the next faulty heating system, and preventing a domestic or occupational disaster in the process.

Other public health concerns involve consideration for the low-level effects of CO in certain, potentially dangerous situations. Given its effects on the central nervous system, one cannot help but wonder how many other injuries or deaths carbon monoxide might actually have a hidden role in, even when a pre-existing disease is not involved. How many fatal traffic accidents, for example, may actually result from drowsiness, nausea, impaired judgment, slowed reflexes, or decreased visual acuity brought on by low to moderate carbon monoxide exposure? Similar questions could be asked about accidents occurring in factories, on construction sites, in homes, or practically anywhere. Although difficult to answer with any authority, questions like these must not be overlooked if the true potential of carbon monoxide’s devastating effects are to be understood.

RECOMMENDATIONS

Happily, accidental carbon monoxide deaths appear to have decreased in frequency over the last half century. The next steps would appear to include more detail demographic studies over a broader area, continued emphasis on public awareness, ongoing suicide prevention programs, and additional public health studies of non-lethal poisonings and their

outcomes. Only through these kinds of efforts will the rates of carbon monoxide morbidity and mortality likely continue to decrease.

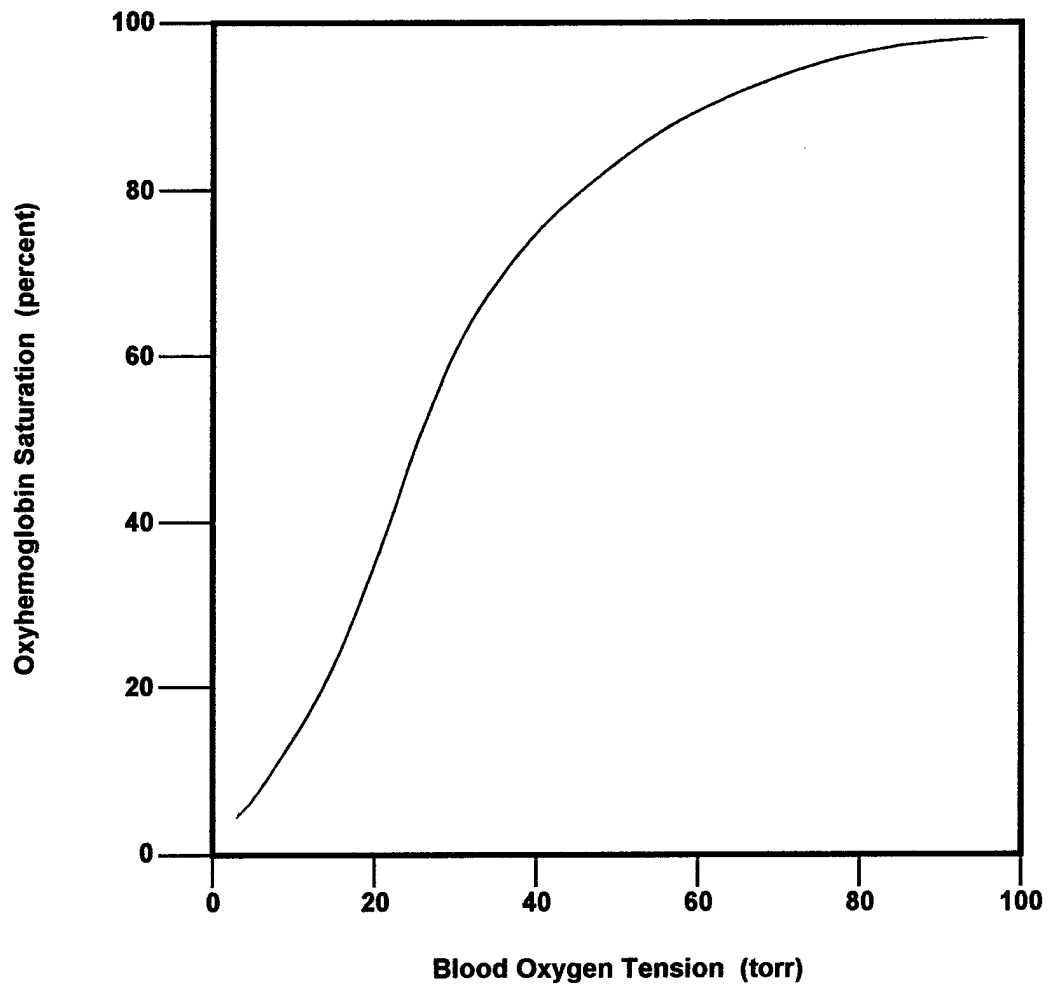
One interesting study would be to assess population "background" blood carboxyhemoglobin levels as a measure of average local, chronic exposure. This could be accomplished through random blood tests, although expense and adequate sampling may be prohibitive. A study involving hospital emergency department data was initially considered for this project, however contact with several San Antonio hospitals led to serious doubts about accurate data retrieval, and further concerns as to completeness of the information. It became apparent that this type of data is not as diligently documented as are trauma cases, and the subjective "chief complaint" in cases such as "nausea," "difficulty breathing," or "dizziness" is often simply repeated as the discharge diagnosis. Obviously, the wide variety of presenting signs and symptoms that can be associated with CO intoxication, coupled with findings that some hospitals' new computer systems could provide only several months of potential data retrieval, prompted me to focus instead on the historical county death data, which was more readily available. Another potentially useful study would involve a survey of the various contractors who test and repair home heating systems. Potential data there might include geographic distribution and demographic information on the help calls, CO levels found in the homes, number of persons in the effected households, and so on.

In any event, this study will perhaps provide at least a starting point for efforts to improving local CO hazard awareness. Hopefully, continued work in this area will help Bexar County's death rates to continue to decline in the decades ahead.

APPENDICIES

APPENDIX A

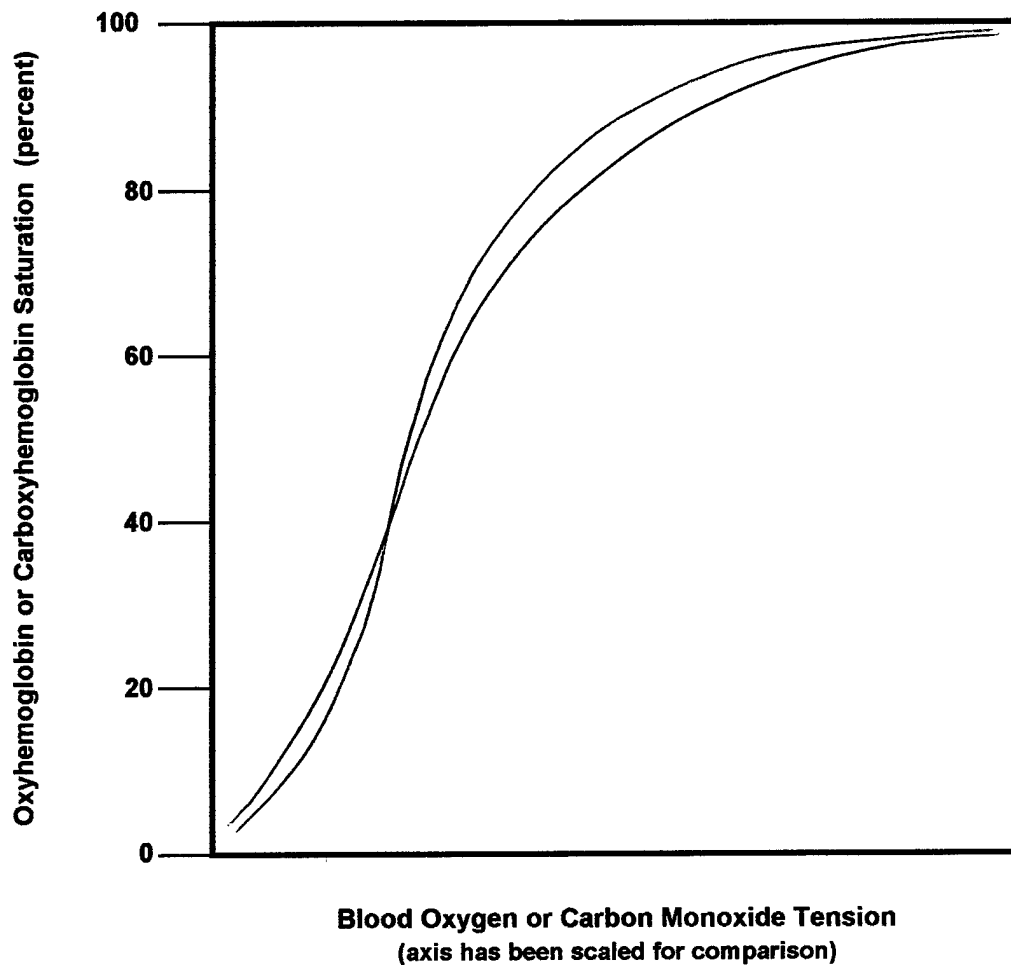
Oxygen dissociation curves at pH 7.4 and 37° C.*



*Adapted from: Ernst A, Zibrak JD. Carbon monoxide poisoning. *N Engl J Med* 1998;339:1603-1608

APPENDIX B

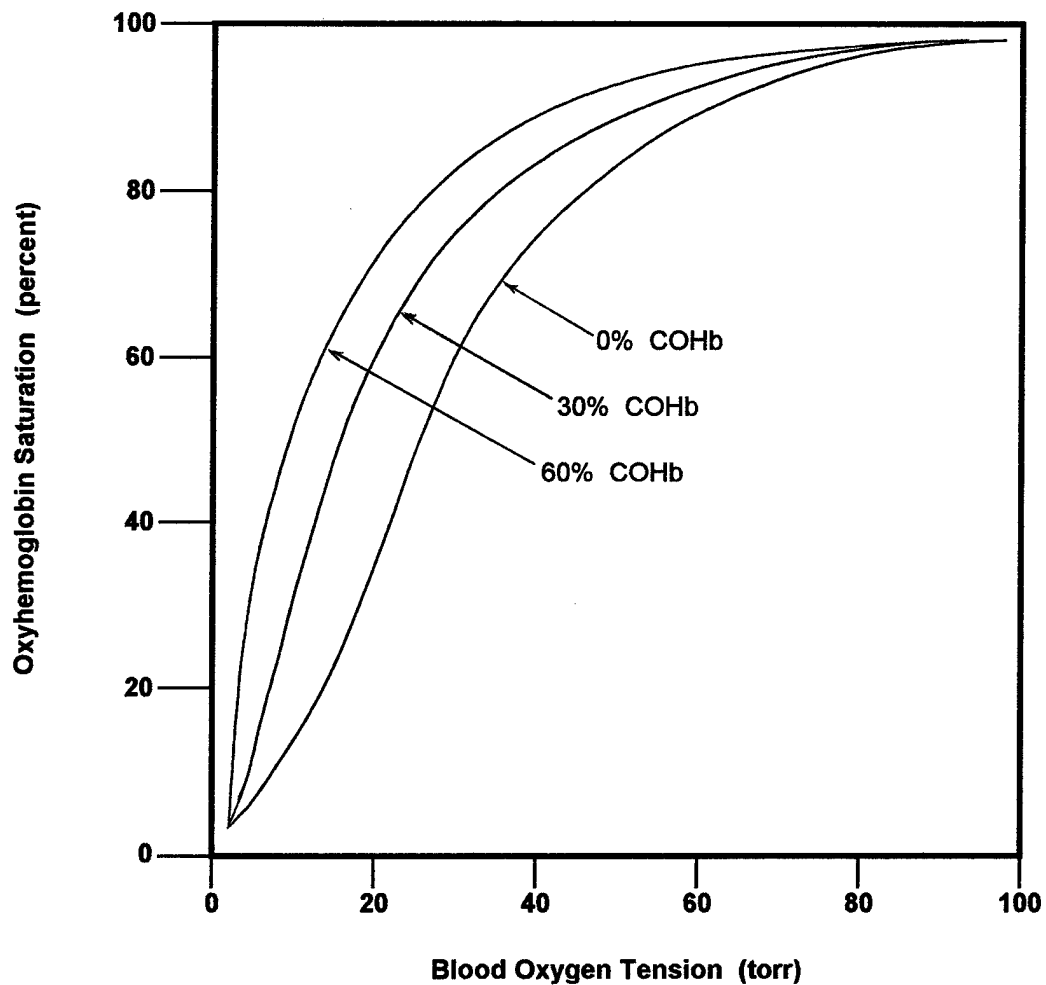
Normalized oxygen (blue) and carbon monoxide (red) dissociation curves (scaled for comparison). Carbon monoxide shows a greater degree of cooperative binding.*



*Adapted from: DiCera E, Doyle ML, Connelly PR, Gill SJ. Carbon monoxide binding to human hemoglobin A₀. *Biochemistry* 1987;26:6494-6502.

APPENDIX C

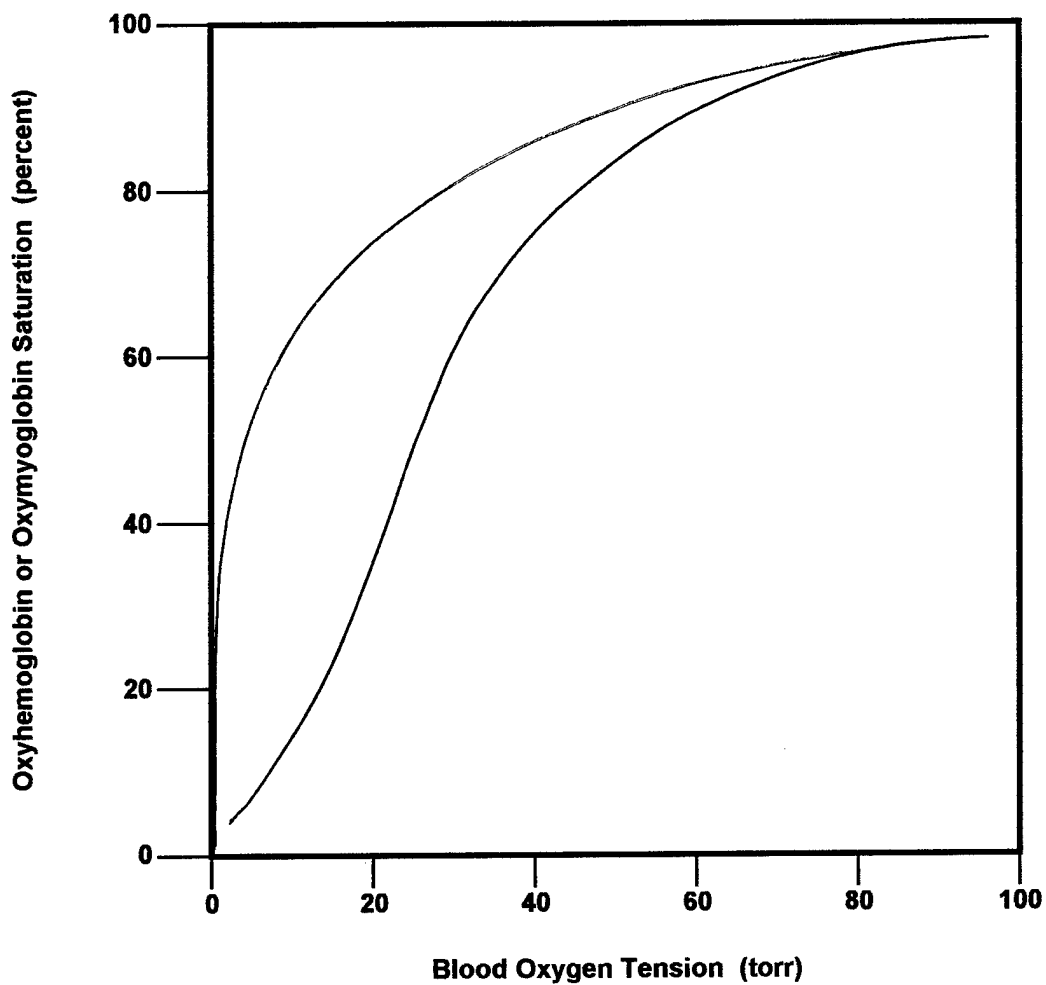
Oxygen dissociation curves at pH 7.4 and 37° C, at varying carboxyhemoglobin saturations. Note that the O₂ saturation curve is shifted to the left as the CO concentration increases.*



*Adapted from: Ernst A, Zibrak JD. Carbon monoxide poisoning. *N Engl J Med* 1998;339:1603-1608.

APPENDIX D

Comparison of oxygen binding to hemoglobin (blue) and myoglobin (green).
The myoglobin curve shows greater saturation at lower oxygen levels, and
lacks cooperative binding.*

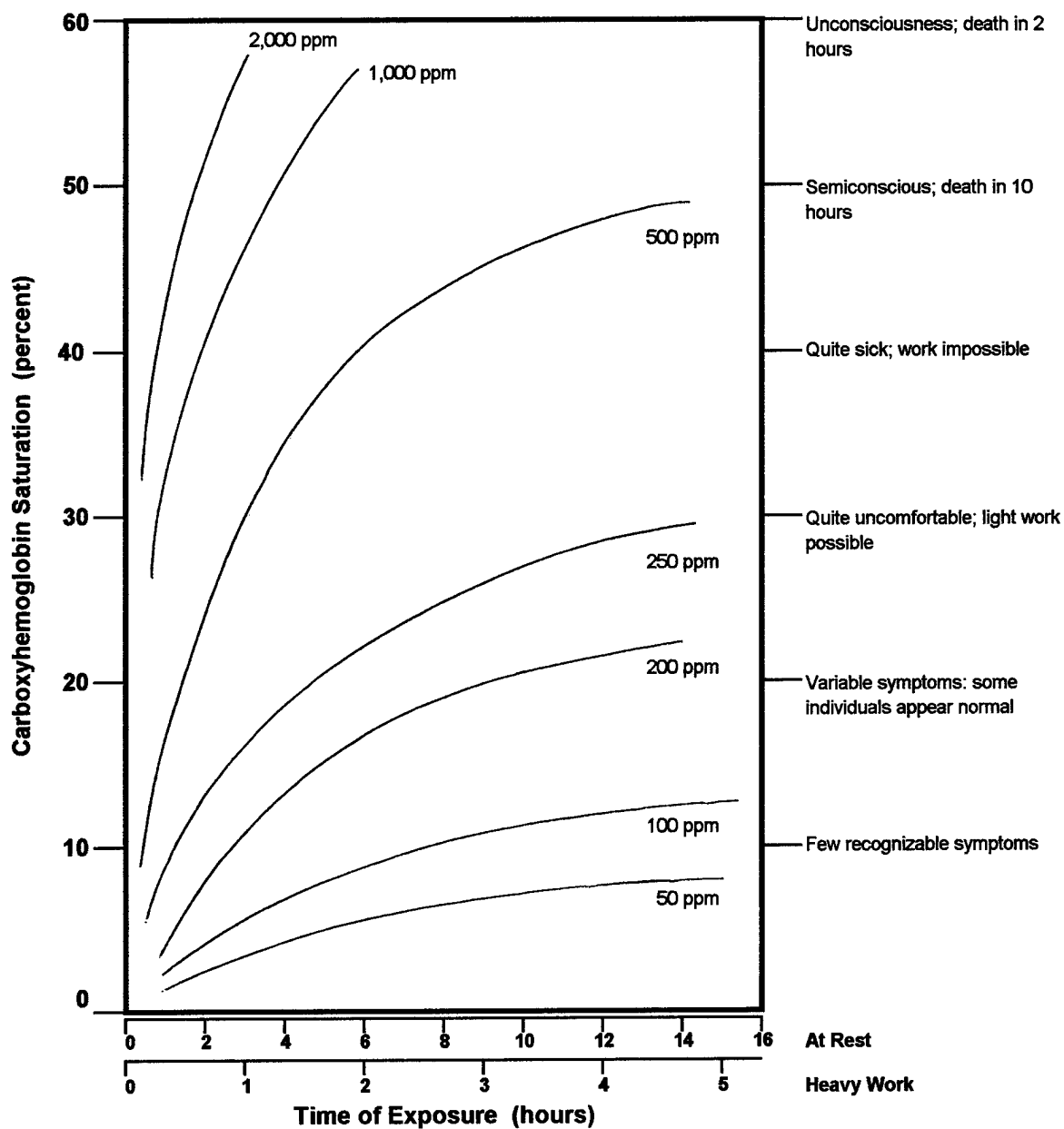


*Adapted from: Alberty RA, Daniels F. *Physical Chemistry*. New York, NY: John Wiley and Sons, 1979.

APPENDIX E

Carboxyhemoglobin saturation as a function of ambient CO concentration, level of physical activity, and exposure time.*

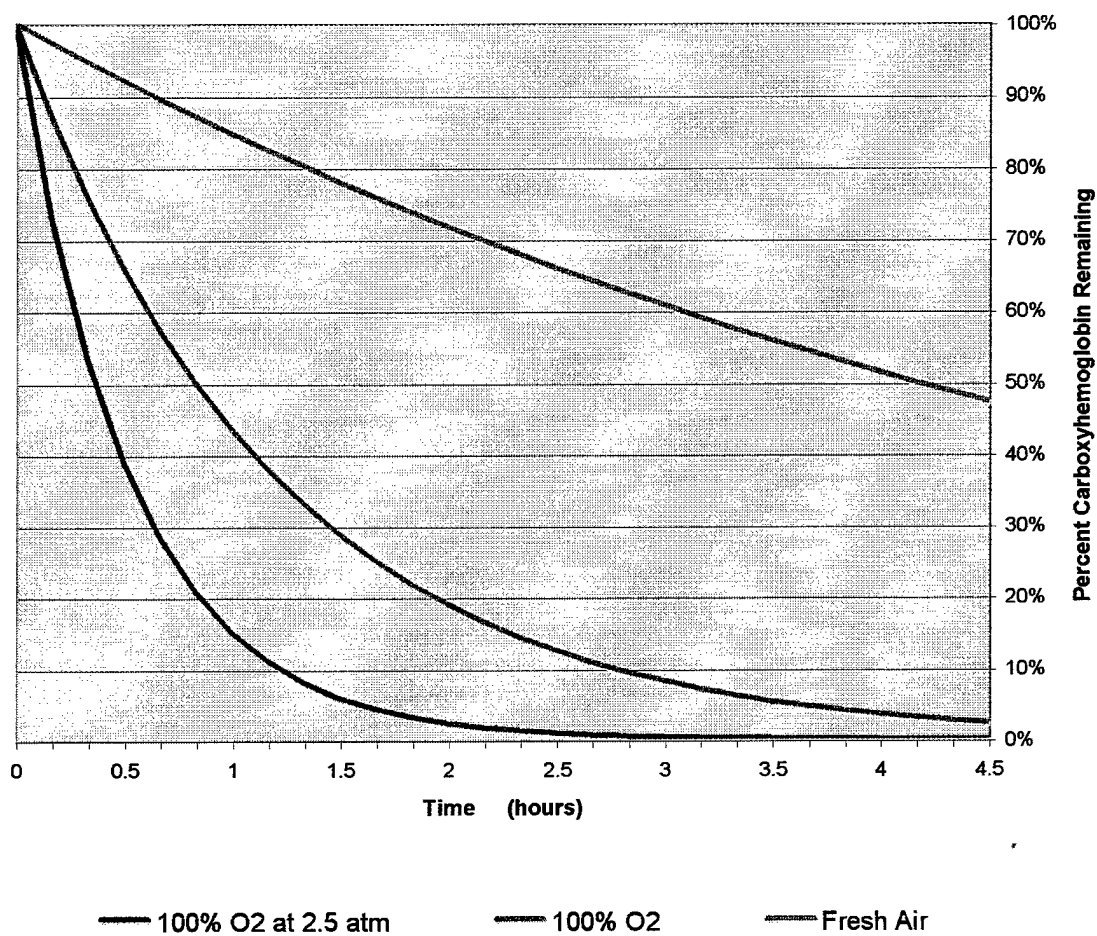
Common Effects:



*Adapted from: Moore JW, Moore EA. *Environmental chemistry*. New York, NY: Academic Press, 1976.
and: Forbes WH. Carbon monoxide uptake via the lungs. *Ann NY Acad Sci* 1970;174:72-75.

APPENDIX F

Rate of carbon monoxide clearance in different oxygen environments



APPENDIX G

ICD-9 codes and descriptions encountered in the 588 cases of CO-related deaths

Code	Text Description
E988.1	Injury by other & unspecified means, undetermined whether accidentally or purposely inflicted. Burns, fire.
E982.9	Poisoning by other gases, undetermined whether accidentally or purposely inflicted. Unspecified gases and vapours.
E982.0	Poisoning by other gases, undetermined whether accidentally or purposely inflicted. Motor vehicle exhaust gas.
E958.9	Suicide and self-inflicted injury by other and unspecified means. Unspecified means.
E953.1	Suicide and self-inflicted injury by hanging, strangulation, and suffocation. Suffocation by plastic bag.
E953.0	Suicide and self-inflicted injury by hanging, strangulation, and suffocation. Hanging.
E952.9	Suicide and self-inflicted poisoning by other gases and vapours. Unspecified gases and vapours.
E952.8	Suicide and self-inflicted poisoning by other gases and vapours. Other specified gases and vapours.
E952.1	Suicide and self-inflicted poisoning by other gases and vapours. Other carbon monoxide.
E952.0	Suicide and self-inflicted poisoning by other gases and vapours. Motor vehicle exhaust gas.
E951.8	Suicide and self-inflicted poisoning by gases in domestic use. Other utility gas.
E951.1	Suicide and self-inflicted poisoning by gases in domestic use. Liquified petroleum gas distributed in mobile containers.
E951.0	Suicide and self-inflicted poisoning by gases in domestic use. Gas distributed by pipeline.
E925.0	Accident caused by electric current. Domestic wiring and appliances.
E923.2	Accident caused by explosive material. Explosive gases.
E900.9	Excessive heat. Of unspecified origin.
E899.0	Accident caused by unspecified fire.
E898.1	Accident caused by other specified fire and flames. Other (blowtorch, candle, cigarette, fire in room, etc.)
E898.0	Accident caused by other specified fire and flames. Burning bedclothes.
E894.0	Ignition of highly inflammable material.
E893.9	Accident caused by ignition of clothing. Unspecified.
E893.8	Accident caused by ignition of clothing. From other sources. (As E893.1 above)
E893.0	Accident caused by ignition of clothing. From controlled fire in private dwelling.
E891.2	Conflagration in other and unspecified building or structure. Other smoke and fumes from conflagration. (Includes CO)
E890.3	Conflagration in private dwelling. Burning caused by conflagration.
E890.2	Conflagration in private dwelling. Other smoke and fumes from conflagration. (Includes CO)
E868.9	Accidental poisoning by other utility gas and other carbon monoxide. Unspecified carbon monoxide.
E868.8	Accidental poisoning by other utility gas and other carbon monoxide. Carbon monoxide from other sources.
E868.3	Accidental poisoning by other utility gas and other CO. Carbon monoxide from incomplete combustion of other domestic fuels.
E868.2	Accidental poisoning by other utility gas and other carbon monoxide. Motor vehicle exhaust gas.
E868.1	Accidental poisoning by other utility gas and other carbon monoxide. Other and unspecified utility gas.
E867.0	Accidental poisoning by distributed by pipeline. Other and unspecified utility gas. (Includes CO from incomplete combustion of piped gas)
E862.1	Accidental poisoning by petroleum products, other solvents, and their vapours, not elsewhere classified. Petroleum fuels and cleaners.
E840.3	Accident to powered aircraft at takeoff or landing. Other occupant. (Non-crew flight personnel, or commercial passenger)
E825.0	Other motor vehicle traffic accident of other and unspecified nature. Driver is person injured. (Includes accidental CO poisoning)
E818.9	Other non-collision motor vehicle traffic accident. Unspecified person. (Includes accidental poisoning from exhaust gas)

Text Description

Code

EB18.0	Other non-collision motor vehicle traffic accident. Driver. (Includes accidental poisoning from exhaust gas)
EB16.1	Motor vehicle traffic accident due to loss of control, without collision on the highway. Passenger.
EB15.0	Other motor vehicle traffic accident involving collision on the highway. Driver.
987.9	Toxic effect of other gases, fumes, or vapours. Unspecified gas.
987.8	Toxic effect of other gases, fumes, or vapours. Other. (Polyester fumes)
987.7	Toxic effect of other gases, fumes, or vapours. Hydrocyanic acid gas.
987.1	Toxic effect of other gases, fumes, or vapours. Other hydrocarbon gases.
987.0	Toxic effect of other gases, fumes, or vapours. Liquified petroleum gases.
986.0	Toxic effect of carbon monoxide. (From all sources)
982.1	Toxic effect of solvents other than petroleum-based. Carbon tetrachloride.
968.0	Poisoning by other central nervous system depressants. CNS muscle-tone depressants.
962.2	Poisoning by hormones and synthetic substitutes. Ovarian hormones and synthetic substitutes.
958.8	Certain early complications of trauma. Other early complications of trauma.
953.1	Injury to nerve roots and spinal plexus. Dorsal root.
953.0	Injury to nerve roots and spinal plexus. Cervical root.
952.1	Spinal cord lesion without evidence of spinal bone injury. Dorsal [thoracic].
952.0	Spinal cord lesion without evidence of spinal bone injury. Cervical.
949.3	Burns, unspecified. Full-thickness skin loss [third degree NOS]
949.2	Burns, unspecified. Blisters, epidermal loss [second degree]
949.0	Burns, unspecified. Unspecified degree.
948.9	Burns classified according to extent of body surface involved. 90% or more.
948.6	Burns classified according to extent of body surface involved. 60-69%.
948.4	Burns classified according to extent of body surface involved. 40-49%.
933.0	Foreign body in pharynx and larynx. Pharynx.
864.0	Injury to liver. Without mention of open wound into cavity.
854.0	Intracranial injury of other and unspecified nature. Without mention of open intracranial wound.
852.0	Subarachnoid, subdural, and extradural haemorrhage, following injury. Without mention of open intracranial wound.
851.0	Cerebral laceration and contusion. Without mention of open intracranial wound.
802.0	Fracture of face bones. Nasal bones, closed.
799.9	Other ill-defined and unknown causes of morbidity and mortality. Other unknown and unspecified cause.
799.3	Other ill-defined and unknown causes of morbidity and mortality. Debility, unspecified.
799.1	Other ill-defined and unknown causes of morbidity and mortality. Respiratory failure.
799.0	Other ill-defined and unknown causes of morbidity and mortality. Asphyxia.
785.4	Symptoms involving cardiovascular system. Gangrene.
780.3	General symptoms. Convulsions.
780.0	General symptoms. Coma and stupor.

Text Description

Code

600.0	Hyperplasia of prostate.
596.3	Other disorders of bladder. Diverticulum of bladder.
590.8	Infections of kidney. Pyelonephritis or pyonephritis, not specified as acute or chronic.
572.8	Liver abscess and sequelae of chronic liver disease. Other sequelae of chronic liver disease.
571.5	Chronic liver disease and cirrhosis. Cirrhosis of liver without mention of alcohol.
514.0	Pulmonary congestion and hypostasis.
491.8	Chronic bronchitis. Other chronic bronchitis.
486.0	Pneumonia, organism unspecified.
485.0	Bronchopneumonia, organism unspecified.
434.9	Occlusion of cerebral arteries. Unspecified.
431.0	Intracranial haemorrhage.
429.2	Ill-defined descriptions and complications of heart disease. Cardiovascular disease, unspecified.
427.5	Cardiac dysrhythmias. Cardiac arrest.
415.1	Acute pulmonary heart disease. Pulmonary embolism.
414.0	Other forms of chronic ischemic heart disease. Coronary atherosclerosis.
410.0	Acute myocardial infarction.
369.0	Blindness and low vision. Blindness, both eyes.
365.9	Glaucoma. Unspecified.
349.9	Other and unspecified disorders of the nervous system. Unspecified.
348.1	Other conditions of brain. Anoxic brain damage.
345.9	Epilepsy. Unspecified.
310.9	Specific nonpsychotic mental disorders following organic brain damage. Unspecified.
306.4	Physiological malfunction arising from mental factors. Gastrointestinal.
305.9	Nondependent abuse of drugs. Other, mixed, or unspecified.
305.1	Nondependent abuse of drugs. Tobacco.
305.0	Nondependent abuse of drugs. Alcohol.
303.0	Alcohol dependence syndrome.
242.9	Thyrotoxicosis with or without goitre. Thyrotoxicosis without mention of goitre or other cause.
199.1	Malignant neoplasm without specification of site. Other. (ie., not disseminated)
153.9	Malignant neoplasm of colon. Colon, unspecified.

APPENDIX H

Abridged database *

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1936	1	61	0	0	1	0	1	8	2	1	1	0	1	sui	Sui Unsp gases
1936	2	59	0	0	1	4	1	8	2	1	9	0	3	sui	Sui Unsp gases
1936	3	43	0	0	1	0	0	8	2	1	1	0	2	sui	Sui MV Exh
1936	4	40	1	0	1	0	1	8	2	1	1	0	24	acc	Acc CO
1936	5	18	1	0	0	3	0	8	2	1	9	0	0	sui	Sui Unsp gases
1937	6	61	0	0	2	0	1	8	2	1	1	0	2	sui	Sui dom gas other
1938	7	31	1	0	2	0	0	8	2	1	1	0	4	sui	Sui Unsp gases
1938	8	21	1	0	0	1	0	8	0	0	1	0	21	acc	Acc util unsp CO
1939	9	36	0	0	0	0	0	8	0	0	9	1	8	acc	Acc util other CO
1939	10	35	0	0	0	4	1	8	0	0	1	0	99	acc	Acc util unsp CO
1939	11	28	1	0	0	0	0	8	2	1	9	1	6	acc	Acc util other CO
1940	12	42	1	0	1	0	0	8	0	8	1	0	24	acc	Acc util unsp CO
1941	13	55	0	0	1	0	0	8	2	8	9	0	3	sui	Sui Unsp gases
1941	14	22	1	0	0	0	0	8	2	8	1	0	0	sui	Sui Unsp gases
1942	15	43	0	0	1	0	0	8	0	8	1	0	9	acc	Acc CO
1943	16	50	1	0	2	0	0	8	2	8	9	0	24	sui	Sui Unsp gases
1944	17	70	0	0	0	0	1	8	2	8	1	0	5	sui	Sui Unsp gases
1944	18	55	1	0	3	0	0	8	2	8	1	0	4	sui	Sui dom gas other
1944	19	21	1	0	1	0	0	8	2	8	1	1	4	acc	Acc util CO dom fuels
1944	20	20	0	0	1	0	0	8	2	8	1	1	42	acc	Acc util CO dom fuels
1945	21	73	0	0	2	3	0	8	0	8	9	0	11	acc	Acc confl CO
1945	22	49	0	0	1	0	1	8	2	8	9	0	4	sui	Sui Unsp gases
1945	23	44	1	0	1	0	0	8	2	8	9	0	24	sui	Sui Unsp gases
1945	24	31	0	0	0	4	1	8	2	8	9	0	42	acc	Acc util unsp CO
1945	25	16	0	0	0	0	0	8	2	8	9	0	0	acc	Acc util unsp CO
1946	26	76	0	0	0	0	0	8	2	8	1	0	1	acc	Acc util unsp CO
1946	27	65	0	0	1	0	1	8	2	8	9	0	3	sui	Sui dom gas by pipe
1946	28	38	0	0	1	3	1	8	2	8	9	0	2	sui	Sui Unsp gases
1946	29	16	1	0	1	3	0	8	0	8	9	0	24	acc	Acc util unsp CO
1947	30	42	0	0	3	9	7	8	2	8	9	0	8	sui	Sui Other CO
1947	31	32	0	0	7	4	7	8	0	8	9	0	98	sui	Sui MV Exh
1948	32	41	0	0	1	0	1	8	2	8	9	0	5	sui	Sui MV Exh
1948	33	22	1	0	1	0	1	8	9	8	9	0	24	sui	Sui Unsp gases
1948	34	1	1	0	0	0	0	8	0	8	9	1	0	acc	Acc MV Exh
1948	35	0	0	0	0	0	0	8	2	8	9	1	0	acc	Acc petrol products
1949	36	66	1	0	2	0	6	8	2	1	9	0	99	sui	Sui Unsp gases
1949	37	50	0	0	1	0	1	8	2	1	1	0	3	sui	Sui MV Exh
1949	38	47	0	0	0	0	0	8	0	1	9	0	3	acc	Acc util CO dom fuels
1949	39	27	1	0	1	0	1	8	3	0	9	0	24	sui	Sui dom gas by pipe
1950	40	56	1	0	2	0	0	8	2	1	9	0	24	sui	Sui dom gas by pipe

* Entire database also listed up to 14 associated causes of death. See APPENDIX I for data codes and column heading descriptions.

** Underlying (primary) cause of death. Although carbon monoxide is not listed in some of the UCD's, all 588 entries have CO toxicity referenced as either the underlying or an associated cause of death.

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1950	41	36	1	0	3	0	0	8	2	9	1	0	4	sui	Sui MV Exh
1950	42	32	1	0	3	0	1	8	2	1	1	0	21	acc	Acc confl CO
1951	43	68	0	0	0	0	6	8	2	9	1	0	5	acc	Acc util unsp CO
1953	44	82	1	0	1	0	0	8	3	1	1	0	24	sui	Sui dom gas by pipe
1953	45	64	0	0	1	0	1	8	0	1	9	0	7	sui	Sui MV Exh
1953	46	57	1	0	0	0	0	8	2	1	1	0	4	sui	Sui Unsp gases
1953	47	50	0	0	1	0	0	8	2	1	1	0	6	sui	Sui MV Exh
1953	48	34	0	0	1	0	0	8	2	1	1	1	8	sui	Sui MV Exh
1954	49	69	0	0	1	0	1	8	2	1	9	0	9	sui	Sui MV Exh
1954	50	63	0	0	1	0	6	8	3	1	1	0	5	sui	Sui MV Exh
1954	51	38	1	0	3	0	1	8	3	1	1	0	4	sui	Sui dom gas by pipe
1954	52	35	1	0	7	0	7	8	3	1	1	0	9	sui	Sui dom gas other
1955	53	62	0	0	0	0	1	8	3	1	1	0	4	sui	Sui MV Exh
1955	54	50	1	0	1	0	0	8	2	1	1	0	24	sui	Sui dom gas by pipe
1955	55	38	0	0	1	0	1	8	2	0	1	0	41	sui	Sui MV Exh
1955	56	29	0	0	0	0	0	8	3	0	1	1	8	sui	Sui MV Exh
1955	57	24	0	0	1	0	0	8	0	1	9	1	8	sui	Sui MV Exh
1956	58	51	0	0	1	0	0	8	0	0	1	0	5	sui	Sui MV Exh
1956	59	41	1	0	1	1	0	8	2	0	1	0	24	sui	Sui dom gas by pipe
1956	60	39	0	1	0	0	0	8	0	0	1	0	8	acc	Acc util unsp CO
1956	61	31	0	0	1	0	0	8	3	0	1	1	4	sui	Sui MV Exh
1956	62	19	1	0	2	0	0	8	2	0	1	0	0	acc	Acc util unsp CO
1956	63	0	0	0	0	0	0	8	0	0	1	1	0	acc	Acc Expl gases
1957	64	96	1	0	0	0	0	8	0	0	1	0	9	acc	Acc confl CO
1957	65	77	0	0	2	0	1	8	3	0	1	0	11	sui	Sui unspc
1957	66	68	1	1	1	0	0	8	3	0	1	0	24	acc	Acc confl CO
1957	67	60	0	0	2	1	0	8	2	0	1	0	3	acc	Acc confl CO
1957	68	55	1	0	1	0	1	8	3	0	0	0	24	sui	Sui dom gas other
1957	69	32	0	0	0	0	0	8	3	0	1	0	3	acc	Acc CO
1957	70	7	0	0	0	0	0	8	3	1	1	1	0	acc	Acc confl CO
1957	71	6	0	0	0	0	0	8	2	1	1	1	0	acc	Acc cntrl fire, dwelling
1957	72	6	1	0	0	0	0	8	3	1	1	1	0	acc	Acc confl CO
1957	73	5	0	0	0	2	0	8	3	1	1	1	0	acc	Acc cntrl fire, dwelling
1957	74	3	0	0	0	0	0	8	2	1	1	1	0	acc	Acc cntrl fire, dwelling
1957	75	2	0	0	0	2	0	8	3	1	1	1	0	acc	Acc cntrl fire, dwelling
1957	76	2	1	0	0	0	0	8	3	1	1	1	0	acc	Acc confl CO
1957	77	2	0	0	0	0	0	8	2	1	1	1	0	acc	Acc cntrl fire, dwelling
1957	78	1	1	0	0	0	0	8	3	1	1	1	0	acc	Acc confl CO
1958	79	88	0	0	2	2	0	8	2	0	1	0	11	acc	Acc confl CO
1958	80	79	1	0	2	0	0	8	2	0	1	0	24	acc	Acc confl CO
1958	81	76	1	0	2	0	1	8	3	0	1	0	24	sui	Sui Other gases
1958	82	67	0	0	1	0	1	8	3	1	1	0	45	acc	Acc confl CO
1958	83	53	1	0	3	0	0	8	2	0	1	0	24	acc	Acc confl CO
1958	84	51	0	1	0	0	0	8	2	0	1	0	8	acc	Acc confl CO
1958	85	50	1	0	2	0	0	8	3	0	1	0	24	acc	Acc confl CO
1958	86	47	0	1	1	0	1	8	2	0	1	0	8	acc	Acc Non-coll MVA
1958	87	44	1	0	1	0	0	8	3	0	1	1	24	sui	Sui dom gas other
1958	88	37	0	0	1	0	0	8	2	0	1	0	3	sui	Sui MV Exh
1958	89	26	0	0	0	1	0	8	3	0	1	0	4	acc	Acc MV Exh
1958	90	17	0	0	0	0	0	8	2	1	1	0	0	sui	Sui dom gas by pipe

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1958	91	2	0	0	0	0	0	8	2	1	1	1	0	acc	Acc confl CO
1959	92	83	0	0	2	0	0	8	0	0	1	1	98	acc	Acc confl CO
1959	93	76	1	0	2	0	0	8	2	0	1	0	24	acc	Acute MI
1959	94	73	1	0	2	2	0	8	3	0	1	0	24	acc	Acc confl CO
1959	95	71	1	0	2	0	1	8	2	0	1	0	24	acc	Acc confl CO
1959	96	66	1	0	3	0	0	8	3	0	1	0	24	acc	Acc confl CO
1959	97	62	0	0	3	0	0	8	3	0	1	0	5	acc	Acc confl CO
1959	98	62	1	0	1	0	0	8	3	0	1	0	24	sui	Sui dom gas other
1959	99	57	0	0	1	2	0	8	0	0	1	0	11	acc	Acc confl CO
1959	100	55	1	0	3	2	0	8	3	0	1	0	1	acc	Acc confl CO
1959	101	51	0	1	3	0	0	8	3	0	1	0	21	acc	Acc confl CO
1959	102	35	1	0	1	0	0	8	3	0	1	0	24	acc	Acc confl CO
1959	103	32	1	0	1	0	0	8	3	0	1	0	4	sui	Sui MV Exh
1959	104	29	0	0	1	0	1	8	2	0	1	0	42	sui	Sui MV Exh
1959	105	18	1	0	1	0	1	8	2	0	0	0	24	sui	Sui dom gas other
1959	106	3	1	1	0	1	0	8	3	0	1	0	0	acc	Acc confl CO
1959	107	1	0	0	0	0	0	8	3	0	1	1	0	acc	Acc confl CO
1959	108	0	1	1	0	1	0	8	3	0	1	0	0	acc	Acc confl CO
1960	109	87	0	0	2	0	2	8	3	0	1	1	98	acc	Acc confl CO
1960	110	65	1	0	1	0	1	8	0	0	1	0	24	sui	Sui Other gases
1960	111	60	0	0	2	2	0	8	2	0	1	0	11	acc	Acc confl CO
1960	112	59	1	0	3	0	1	8	3	1	1	0	24	sui	Sui dom gas by pipe
1960	113	54	1	0	3	0	0	8	3	1	1	0	4	acc	Acc electric dom
1960	114	52	0	1	0	0	1	8	3	0	1	0	8	acc	Acc confl CO
1960	115	50	1	0	1	0	0	8	3	1	1	1	24	sui	Sui dom gas by pipe
1960	116	49	0	0	1	0	0	8	3	0	1	1	5	acc	Acc confl CO
1960	117	43	0	0	1	0	2	8	3	0	0	1	7	acc	Acc MVA w/ CO
1960	118	43	1	0	1	0	1	8	2	1	1	0	24	sui	Sui MV Exh
1960	119	42	1	0	0	1	1	8	3	0	1	0	1	acc	Acc confl CO
1960	120	22	0	0	0	0	0	8	3	1	1	0	4	sui	Sui dom gas by pipe
1960	121	20	0	0	1	0	0	8	0	0	1	1	6	acc	Acc confl CO
1960	122	1	0	1	0	0	0	8	3	0	1	0	0	acc	Acc confl CO
1961	123	79	0	0	2	0	2	8	3	0	1	1	8	acc	Acc fire other
1961	124	65	0	0	3	0	1	8	3	0	1	0	2	acc	Acc confl CO
1961	125	59	1	0	2	0	0	8	2	0	1	0	24	sui	Sui dom gas other
1961	126	55	1	0	1	0	1	8	3	0	1	0	24	acc	Acc confl CO
1961	127	52	0	0	1	0	1	8	2	0	1	0	5	sui	Sui Other gases
1961	128	48	0	0	1	0	0	8	3	0	0	0	98	sui	Sui MV Exh
1961	129	42	0	0	1	4	1	8	3	0	0	0	7	acc	Acc Non-coll MVA
1961	130	42	0	0	3	1	1	8	3	1	1	0	41	sui	Sui Other CO
1961	131	35	0	0	1	0	1	8	3	0	1	0	41	sui	Sui MV Exh
1961	132	31	0	0	1	0	1	8	3	0	1	0	6	sui	Sui MV Exh
1961	133	26	0	0	3	2	1	8	3	0	1	0	5	acc	Acc confl CO
1961	134	26	0	0	0	0	0	8	3	0	1	0	2	sui	Sui dom gas by pipe
1961	135	26	1	0	1	3	0	8	0	0	1	0	24	acc	Acc CO
1961	136	25	0	0	1	0	1	8	3	0	1	1	5	acc	Acc CO
1961	137	1	1	0	0	0	0	8	0	0	9	0	0	acc	Acc confl CO
1961	138	0	0	0	0	0	0	8	0	0	1	0	0	acc	Acc confl CO
1962	139	90	0	0	3	2	1	8	2	0	1	0	98	acc	Acc confl CO
1962	140	79	1	0	2	0	0	8	3	1	1	0	24	sui	Sui dom gas liq petr

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1962	141	67	0	0	3	0	1	8	3	0	1	0	1	acc	Acc util other CO
1962	142	63	0	0	0	0	1	8	3	0	1	0	42	sui	Sui MV Exh
1962	143	61	1	0	2	0	0	8	3	0	1	0	24	sui	Sui dom gas by pipe
1962	144	59	0	0	1	0	1	8	3	0	1	0	4	sui	Sui MV Exh
1962	145	57	0	0	2	1	1	8	2	0	9	0	2	acc	Acc MV Exh
1962	146	54	1	0	1	0	1	8	3	0	1	0	24	sui	Sui dom gas other
1962	147	52	0	0	0	0	0	8	3	0	1	1	8	acc	Acc confl CO
1962	148	52	1	0	1	0	0	8	2	0	1	0	24	sui	Sui Other CO
1962	149	51	1	0	0	0	1	8	2	0	1	0	2	acc	Acc confl CO
1962	150	49	1	0	1	1	1	8	2	0	9	0	4	sui	Sui MV Exh
1962	151	46	0	0	1	0	1	8	3	0	1	0	5	sui	Sui MV Exh
1962	152	45	0	0	3	0	1	8	3	0	9	0	42	sui	Sui MV Exh
1962	153	42	1	0	1	0	1	8	3	0	1	0	3	acc	Acc ig of flamables
1962	154	41	0	1	1	0	0	8	3	0	1	0	98	acc	Acc confl CO
1962	155	33	1	0	3	0	0	8	2	0	1	0	4	sui	Sui dom gas other
1962	156	29	0	0	1	0	0	8	3	1	1	1	4	sui	Sui Other CO
1962	157	2	0	0	0	0	0	8	3	1	1	1	0	acc	Acc ig of flamables
1962	158	2	1	0	0	0	1	8	3	0	1	0	0	acc	Acc heat unsp
1963	159	87	1	0	2	0	1	8	3	0	1	0	24	acc	Acc burned clothing
1963	160	82	0	0	1	0	7	8	3	1	1	0	4	acc	Acc confl CO
1963	161	73	0	0	1	0	0	8	3	0	1	0	8	acc	Acc confl CO
1963	162	67	0	0	2	0	1	8	2	0	1	0	1	acc	Acc confl CO
1963	163	58	1	0	2	0	1	8	3	1	1	0	3	sui	Sui dom gas other
1963	164	55	1	0	3	0	1	8	3	0	1	0	4	sui	Sui dom gas other
1963	165	52	1	0	1	0	2	8	3	0	1	1	24	sui	Sui dom gas other
1963	166	51	1	0	2	0	0	8	0	0	1	0	4	acc	Acc ig of clothing
1963	167	41	0	0	1	0	0	8	3	0	1	0	3	sui	Sui MV Exh
1963	168	41	1	0	1	0	0	8	0	1	1	1	24	sui	Sui dom gas other
1963	169	37	1	0	1	0	0	8	3	1	1	0	4	sui	Sui dom gas other
1963	170	36	1	0	1	1	0	8	0	1	1	0	24	sui	Sui dom gas other
1963	171	34	0	0	0	4	0	8	3	1	1	0	1	sui	Sui MV Exh
1963	172	32	1	0	1	0	0	8	3	0	9	0	9	acc	Acc ig of clothing
1963	173	26	0	0	1	0	2	8	3	1	1	1	9	sui	Sui dom gas other
1963	174	25	0	0	0	0	0	8	0	0	1	1	9	sui	Sui dom gas other
1963	175	19	1	0	1	0	0	8	3	0	1	0	24	sui	Sui Suff
1963	176	16	0	1	0	0	0	8	3	0	1	0	0	sui	Sui dom gas by pipe
1963	177	1	1	0	0	0	0	8	3	0	1	1	0	acc	Acc confl CO
1964	178	68	0	0	2	1	1	8	2	1	1	0	1	sui	Sui MV Exh
1964	179	67	0	0	3	2	0	8	3	0	1	0	8	acc	Acc confl burns
1964	180	65	1	0	1	1	0	8	3	1	1	0	24	sui	Sui MV Exh
1964	181	63	0	0	2	0	1	8	3	1	1	0	41	sui	Sui dom gas by pipe
1964	182	53	0	0	1	0	0	8	3	1	1	0	3	sui	Sui MV Exh
1964	183	47	0	0	1	0	0	8	3	9	1	0	7	acc	Acc fire other
1964	184	45	1	1	3	1	0	8	3	0	1	0	21	sui	Sui MV Exh
1964	185	32	0	0	0	2	1	8	3	0	1	0	41	sui	Sui MV Exh
1965	186	73	0	0	2	2	0	8	3	1	1	0	12	sui	Sui MV Exh
1965	187	59	0	0	0	0	0	8	3	0	1	0	7	acc	Acc bedclothes fire
1965	188	56	0	0	1	0	0	8	3	0	1	0	2	sui	Sui dom gas liq petr
1965	189	55	0	0	1	0	1	8	3	0	1	0	72	acc	Acc cntrl fire, dwelling
1965	190	44	0	0	1	0	0	8	3	0	1	0	5	acc	Acc util CO dom fuels

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1965	191	44	0	0	1	1	2	8	3	0	1	0	2	acc	Acc confl CO
1965	192	38	1	0	2	0	1	8	2	0	1	0	24	sui	Sui MV Exh
1965	193	25	1	0	1	0	2	8	0	0	1	1	24	sui	Sui dom gas other
1965	194	24	1	0	0	0	0	8	3	0	1	0	1	sui	Sui MV Exh
1965	195	21	0	0	0	0	0	8	3	0	1	1	6	acc	Acc MV Exh
1965	196	21	0	0	0	0	0	8	3	1	1	0	4	sui	Sui dom gas other
1965	197	16	0	1	0	0	0	8	3	0	1	0	0	sui	Sui dom gas other
1966	198	77	1	0	1	0	0	8	3	1	9	0	1	acc	Acc util other CO
1966	199	66	1	0	1	0	0	8	3	1	1	0	24	sui	Sui dom gas other
1966	200	64	1	0	1	2	0	8	1	0	1	0	24	acc	Acc MVA
1966	201	57	0	0	1	0	1	8	3	0	1	0	3	sui	Sui dom gas other
1966	202	57	0	0	1	0	0	8	3	1	1	0	4	sui	Sui MV Exh
1966	203	54	1	0	3	0	0	8	2	1	1	0	4	sui	Sui dom gas other
1966	204	53	1	0	3	0	0	8	2	0	1	0	24	sui	Sui dom gas other
1966	205	52	0	0	1	0	1	8	3	1	1	0	4	sui	Sui MV Exh
1966	206	51	1	0	1	0	1	8	3	0	1	1	4	sui	Sui MV Exh
1966	207	51	0	0	1	0	1	8	3	1	1	0	4	sui	Sui MV Exh
1966	208	46	1	0	0	0	0	8	3	1	1	0	4	sui	Sui dom gas other
1966	209	46	0	0	1	1	1	8	3	0	1	0	45	und	UND MV Ex
1966	210	44	1	0	1	0	0	8	3	1	1	0	24	sui	Sui MV Exh
1966	211	41	1	0	1	0	0	8	2	1	1	0	2	sui	Sui dom gas other
1966	212	39	1	1	1	0	0	8	3	0	1	0	4	sui	Sui dom gas other
1966	213	36	0	0	1	0	1	8	0	0	1	0	7	sui	Sui Hanging
1966	214	34	0	0	3	0	0	8	3	1	1	0	5	sui	Sui dom gas other
1966	215	32	0	0	1	0	0	8	0	1	1	0	2	acc	Acc confl burns
1966	216	31	0	0	3	0	0	8	3	1	1	0	5	sui	Sui MV Exh
1966	217	29	1	0	1	0	1	8	3	1	1	0	9	sui	Sui MV Exh
1966	218	28	1	0	3	0	0	8	3	0	1	0	4	acc	Acc confl burns
1966	219	22	0	0	0	0	0	8	3	0	1	0	0	sui	Sui MV Exh
1966	220	21	0	0	0	0	0	8	0	1	1	1	6	sui	Sui MV Exh
1966	221	21	1	1	1	0	0	8	3	0	1	0	24	sui	Sui dom gas other
1966	222	19	1	1	0	0	0	8	3	1	1	0	21	sui	Sui dom gas other
1966	223	17	0	0	0	0	0	8	3	1	1	0	0	sui	Sui MV Exh
1966	224	7	0	0	0	0	0	8	3	0	1	0	0	acc	Acc util other CO
1967	225	71	0	0	3	2	0	8	3	1	1	0	11	acc	Acc util CO dom fuels
1967	226	69	0	0	1	0	0	8	3	1	1	0	3	sui	Sui dom gas other
1967	227	66	0	0	1	1	1	8	3	1	1	0	2	sui	Sui MV Exh
1967	228	65	1	0	2	1	0	8	2	1	1	0	24	sui	Sui dom gas other
1967	229	64	0	0	1	0	2	8	3	0	1	1	5	acc	Acc confl CO
1967	230	62	0	1	2	0	0	8	3	0	1	0	9	acc	Acc confl CO
1967	231	62	0	0	1	0	1	8	3	0	1	0	1	sui	Sui MV Exh
1967	232	60	0	0	7	7	7	8	2	0	1	0	99	acc	Acc confl CO
1967	233	59	1	1	1	0	0	8	3	0	1	0	22	acc	Alcohol dep synd
1967	234	51	1	1	1	0	0	8	3	0	1	0	9	acc	Acc util unsp CO
1967	235	42	0	0	3	0	0	8	3	0	1	1	7	acc	Acc confl burns
1967	236	41	0	0	2	0	1	8	3	0	1	0	41	sui	Sui MV Exh
1967	237	38	0	0	1	0	0	8	3	0	1	1	5	acc	Acc MV Exh
1967	238	34	0	0	1	0	1	8	3	0	1	0	42	acc	Acc confl CO
1967	239	34	0	0	1	0	2	8	3	1	1	1	3	sui	Sui MV Exh
1967	240	32	1	0	1	2	1	8	3	1	1	0	24	sui	Sui MV Exh

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1967	241	31	0	0	0	0	0	8	0	0	1	1	9	acc	Acc util unsp CO
1967	242	20	0	0	0	0	0	8	3	0	1	1	44	acc	Acc Non-coll MVA
1967	243	20	0	0	0	0	1	8	2	1	1	0	0	sui	Sui MV Exh
1967	244	18	1	0	0	0	0	8	3	0	1	1	0	acc	Acc Non-coll MVA
1967	245	3	0	0	0	0	0	8	3	1	1	1	0	acc	Acc confl burns
1967	246	2	1	1	0	0	0	8	2	0	1	0	0	acc	Acc confl burns
1967	247	0	0	1	0	0	0	8	2	0	1	0	0	acc	Acc confl burns
1968	248	82	1	0	0	0	0	8	3	1	1	0	1	acc	Acc confl CO
1968	249	80	1	0	2	0	0	8	3	0	7	0	1	und	UND MV Ex
1968	250	79	0	0	0	0	2	8	0	0	1	1	8	acc	Acc CO
1968	251	78	1	0	0	0	0	8	3	1	1	0	1	acc	Acc confl CO
1968	252	75	1	0	0	0	0	8	3	0	1	0	1	acc	Acc confl CO
1968	253	68	0	0	1	2	6	8	2	0	9	0	8	acc	Acc confl CO
1968	254	56	0	0	1	0	2	8	3	1	1	1	5	sui	Sui MV Exh
1968	255	56	0	0	3	0	1	8	3	0	1	0	8	acc	Acc fire unsp
1968	256	56	0	0	0	3	0	8	3	0	1	0	11	acc	Acc fire other
1968	257	54	0	0	1	0	0	8	0	0	1	0	7	acc	Acc MV Exh
1968	258	49	1	0	2	0	0	8	3	0	1	0	24	acc	Acc confl CO
1968	259	46	0	0	1	0	1	8	2	0	0	0	2	acc	Acc Aircraft
1968	260	38	1	0	1	0	1	8	3	1	1	0	24	sui	Sui MV Exh
1968	261	30	0	0	0	2	0	8	3	1	1	0	4	sui	Sui MV Exh
1968	262	27	1	0	1	0	1	8	3	0	1	0	24	acc	Acc burned clothing
1968	263	25	1	0	0	0	0	8	0	0	1	0	1	sui	Sui MV Exh
1968	264	21	1	0	0	0	0	8	3	1	1	0	0	acc	Acc confl CO
1968	265	3	1	1	0	2	0	8	3	0	1	0	0	acc	Acc confl CO
1968	266	0	0	0	0	2	0	8	2	0	1	0	0	acc	Acc confl CO
1969	267	72	0	0	2	0	1	8	3	1	1	0	1	acc	Acc MV Exh
1969	268	57	0	0	1	0	1	8	2	0	1	0	3	acc	Acc confl CO
1969	269	53	1	0	1	0	0	8	2	0	1	0	24	acc	Acc confl CO
1969	270	49	1	0	1	0	0	8	3	0	1	0	1	sui	Sui MV Exh
1969	271	48	0	0	1	2	1	8	3	0	1	0	41	acc	Acc fire other
1969	272	35	1	0	1	2	0	8	3	1	9	0	24	acc	Acc confl CO
1969	273	28	0	1	0	0	0	8	3	0	1	0	8	acc	Acc confl CO
1969	274	28	1	0	1	2	1	8	3	1	1	0	24	sui	Sui MV Exh
1969	275	27	1	0	1	0	0	8	3	0	1	1	24	sui	Sui dom gas other
1969	276	26	0	0	1	0	0	8	3	1	1	0	0	sui	Sui Suff
1969	277	21	0	0	0	0	0	8	3	0	1	0	4	sui	Sui MV Exh
1969	278	19	0	0	3	0	0	8	3	0	1	0	44	sui	Sui MV Exh
1969	279	17	0	0	0	2	0	8	3	1	1	0	0	acc	Acc confl CO
1969	280	16	1	0	0	2	0	8	3	1	1	0	0	acc	Acc confl CO
1969	281	15	1	0	0	2	0	8	3	1	1	0	0	acc	Acc confl CO
1969	282	12	0	0	0	2	0	8	3	1	1	0	0	acc	Acc confl CO
1969	283	5	1	0	0	2	0	8	3	1	1	0	0	acc	Acc confl CO
1970	284	81	1	0	2	0	2	8	0	1	1	1	24	acc	Acc ig of clothing
1970	285	71	1	0	2	0	1	8	3	0	1	0	24	acc	Acc util unsp CO
1970	286	66	0	0	1	0	1	8	3	1	1	0	98	sui	Sui MV Exh
1970	287	51	1	0	1	0	1	8	3	0	1	0	24	sui	Sui MV Exh
1970	288	41	1	0	3	2	1	8	3	0	1	0	1	acc	Acc confl burns
1970	289	32	0	0	1	0	0	8	3	1	1	0	5	sui	Sui MV Exh
1970	290	27	0	0	7	7	7	8	3	1	1	0	98	sui	Sui MV Exh

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1970	291	23	0	0	1	0	2	8	0	0	9	1	6	und	Thyrototoxicosis
1970	292	21	0	1	0	0	0	8	2	1	1	0	8	acc	Acc util unsp CO
1970	293	20	0	0	0	0	0	8	2	0	1	0	0	acc	Acc util CO dom fuels
1970	294	20	0	0	0	3	6	8	3	0	1	0	0	acc	Acc other MVA
1971	295	77	1	0	2	0	1	8	0	1	1	0	24	acc	Acc CO
1971	296	44	0	0	1	0	0	8	3	1	1	0	98	sui	Sui MV Exh
1971	297	42	0	0	3	0	0	8	0	0	1	1	5	acc	Acc ig of clothing
1971	298	42	0	0	1	1	1	8	0	1	1	0	3	sui	Sui MV Exh
1971	299	42	1	0	1	0	0	8	3	0	9	0	24	sui	Sui MV Exh
1971	300	41	1	0	1	0	0	8	3	1	1	0	24	sui	Sui MV Exh
1971	301	40	1	0	1	0	1	8	3	1	1	0	24	sui	Sui MV Exh
1971	302	39	1	0	1	2	0	8	0	1	9	0	3	sui	Sui MV Exh
1971	303	34	1	0	1	0	1	8	3	1	1	0	24	sui	Sui MV Exh
1971	304	32	0	0	1	2	0	8	3	1	1	0	2	sui	Sui MV Exh
1971	305	29	1	0	3	0	1	8	3	1	1	0	9	acc	Acc confl CO
1971	306	29	1	0	2	0	0	8	3	1	1	0	4	sui	Sui MV Exh
1971	307	21	0	0	1	2	0	8	0	0	9	1	42	acc	Acc util unsp CO
1971	308	18	1	0	1	0	0	8	0	0	1	1	24	acc	Acc util unsp CO
1971	309	17	0	0	0	2	0	8	3	1	1	0	0	acc	Acc MV Exh
1971	310	13	1	1	0	0	0	8	3	1	1	0	0	acc	Acc confl burns
1971	311	7	1	0	0	0	1	8	3	1	1	0	0	acc	Acc confl CO
1971	312	4	0	0	0	0	1	8	3	1	1	0	0	acc	Acc confl CO
1972	313	75	0	0	1	0	0	8	3	1	1	0	5	sui	Sui MV Exh
1972	314	75	1	0	2	0	0	8	3	1	1	0	9	acc	Acc Non-coll MVA
1972	315	73	0	0	2	0	1	8	3	1	1	0	1	acc	Acc confl CO
1972	316	72	0	0	2	0	1	8	3	1	1	0	41	sui	Sui MV Exh
1972	317	71	0	0	3	0	2	8	3	1	1	1	8	acc	Acc CO
1972	318	60	1	0	2	0	1	8	3	1	1	0	24	sui	Sui MV Exh
1972	319	54	1	0	1	0	1	8	3	1	1	0	24	sui	Sui MV Exh
1972	320	50	0	0	3	0	0	8	3	1	1	0	98	acc	Acc confl CO
1972	321	42	0	1	3	0	1	8	3	1	1	0	98	acc	Acc confl CO
1972	322	32	0	0	0	0	0	8	3	1	1	1	8	acc	Acc CO
1972	323	25	0	0	0	3	0	8	3	1	1	0	0	acc	Acc CO
1972	324	25	0	0	1	0	0	8	3	1	1	1	8	sui	Sui MV Exh
1972	325	6	0	1	0	0	0	8	3	1	1	0	0	acc	Acc confl burns
1972	326	0	0	0	0	0	0	8	0	0	1	0	0	acc	Acc CO
1973	327	80	1	0	2	0	0	8	0	1	1	0	24	acc	Acc confl burns
1973	328	75	0	0	2	0	1	8	3	0	1	0	4	acc	Acc confl CO
1973	329	57	1	0	1	0	0	8	3	0	1	0	4	sui	Sui MV Exh
1973	330	54	0	0	3	0	0	8	3	1	1	1	1	sui	Sui dom gas by pipe
1973	331	52	0	0	2	0	0	8	3	0	1	0	41	sui	Sui MV Exh
1973	332	49	0	0	1	0	1	8	3	1	1	0	2	sui	Sui MV Exh
1973	333	49	0	0	1	0	0	8	3	1	1	1	7	sui	Sui MV Exh
1973	334	48	1	0	1	2	0	8	3	1	1	0	24	sui	Sui MV Exh
1973	335	48	1	0	1	0	1	8	3	0	1	0	24	sui	Sui MV Exh
1973	336	46	0	0	2	0	0	8	3	1	1	0	1	sui	Sui MV Exh
1973	337	44	0	0	1	0	0	8	3	0	1	0	42	acc	Acc util unsp CO
1973	338	38	0	0	1	0	1	8	3	9	1	0	1	sui	Sui MV Exh
1973	339	36	1	0	2	1	0	8	3	1	1	0	1	sui	Sui MV Exh
1973	340	33	0	0	0	0	0	8	3	1	1	0	0	acc	Acc confl CO

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1973	341	28	1	0	3	0	1	8	3	0	1	0	9	acc	Acc Confl sm/fumes
1973	342	23	0	0	3	0	0	8	3	1	1	0	5	sui	Sui MV Exh
1973	343	16	1	0	0	0	1	8	3	1	1	0	0	acc	Acc util CO dom fuels
1973	344	13	1	0	0	0	1	8	3	1	1	0	0	acc	Acc util CO dom fuels
1974	345	67	0	0	0	0	2	8	3	1	1	0	8	acc	Acc confl CO
1974	346	65	0	0	1	0	1	8	3	1	1	0	3	sui	Sui dom gas by pipe
1974	347	55	0	0	1	0	1	8	3	1	1	0	41	sui	Sui MV Exh
1974	348	50	0	0	3	0	0	8	3	1	9	0	3	sui	Sui MV Exh
1974	349	47	0	0	1	1	0	8	3	0	1	0	2	sui	Sui MV Exh
1974	350	9	0	1	0	0	0	8	0	1	1	0	0	acc	Acc confl CO
1975	351	66	0	0	2	0	0	8	3	1	1	1	8	acc	Acc confl CO
1975	352	63	1	0	1	0	0	8	3	1	1	0	24	sui	Sui dom gas by pipe
1975	353	59	1	0	1	0	0	8	3	1	1	0	24	acc	Acc confl CO
1975	354	59	0	0	1	0	1	8	3	1	1	0	1	sui	Sui MV Exh
1975	355	58	0	0	2	2	0	8	3	1	1	0	2	sui	Sui MV Exh
1975	356	55	0	0	1	3	1	8	3	1	9	0	5	sui	Sui MV Exh
1975	357	54	0	0	1	1	0	8	3	1	1	0	1	acc	Acc MV Exh
1975	358	51	0	0	3	0	0	8	0	1	1	0	6	acc	Acc bedclothes fire
1975	359	44	0	0	3	0	1	8	3	1	1	0	5	und	UND MV Ex
1975	360	32	0	0	3	0	1	8	3	1	1	0	5	sui	Sui MV Exh
1975	361	32	1	0	3	0	1	8	3	0	1	0	4	sui	Sui MV Exh
1975	362	31	0	0	0	5	2	8	3	1	1	1	12	acc	Acc CO
1975	363	28	1	0	1	0	0	8	3	1	1	0	4	sui	Sui MV Exh
1975	364	27	0	0	1	4	1	8	3	1	1	0	42	sui	Sui MV Exh
1975	365	26	0	0	1	0	0	8	3	1	1	0	3	sui	Sui MV Exh
1975	366	25	0	0	0	0	1	8	0	0	1	0	41	acc	Pneumonia
1975	367	24	0	0	1	5	2	8	3	1	1	1	12	acc	Acc CO
1976	368	76	0	0	1	1	0	8	3	0	1	0	3	acc	Acc confl CO
1976	369	64	1	0	2	1	0	8	3	0	1	0	24	sui	Sui MV Exh
1976	370	47	0	0	1	0	0	8	3	1	1	0	5	sui	Sui dom gas by pipe
1976	371	46	0	0	1	0	0	8	3	1	1	0	42	sui	Sui MV Exh
1976	372	45	1	0	3	1	0	8	3	1	9	0	2	sui	Sui MV Exh
1976	373	42	0	0	1	2	1	8	3	1	1	0	41	sui	Sui MV Exh
1976	374	39	0	0	1	0	1	8	0	0	9	0	41	und	UND MV Ex
1976	375	39	1	0	1	1	1	8	3	1	1	0	24	sui	Sui MV Exh
1976	376	36	1	1	9	0	0	8	3	0	9	0	1	und	UND MV Ex
1976	377	29	0	0	0	0	6	8	3	0	1	0	5	acc	Acc confl burns
1976	378	24	1	0	0	1	0	8	3	0	1	0	1	acc	Acc confl CO
1976	379	20	0	0	1	0	1	8	3	1	1	0	42	acc	Acc CO
1976	380	19	1	0	1	0	1	8	3	1	1	0	24	acc	Acc CO
1976	381	19	0	0	1	0	0	8	3	1	1	1	5	acc	Acc CO
1976	382	18	1	0	1	0	0	8	3	1	1	1	0	acc	Acc CO
1976	383	16	0	0	0	0	0	8	3	1	1	0	0	sui	Sui Hanging
1976	384	11	0	0	0	0	0	8	3	1	1	0	0	acc	Acc confl burns
1977	385	84	0	0	1	0	0	8	3	1	1	0	11	sui	Sui MV Exh
1977	386	70	1	0	2	0	0	8	3	1	1	0	24	sui	Sui MV Exh
1977	387	67	1	0	2	0	0	8	3	0	1	0	5	acc	Acc confl CO
1977	388	67	0	0	1	0	2	8	3	1	9	1	98	acc	Acc util CO dom fuels
1977	389	61	0	0	1	0	0	8	3	1	0	0	9	acc	Acc CO
1977	390	56	0	0	1	0	0	8	3	1	9	0	5	acc	Acc MV Exh

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1977	391	39	0	0	3	1	1	8	3	1	1	0	3	sui	Sui MV Exh
1977	392	39	0	0	1	0	1	8	3	0	0	0	42	sui	Sui MV Exh
1977	393	28	1	0	1	0	1	8	3	1	1	0	24	sui	Sui MV Exh
1977	394	22	0	0	0	0	0	8	3	1	1	0	0	sui	Sui MV Exh
1978	395	93	1	0	2	1	0	8	3	1	1	0	1	acc	Acc confl CO
1978	396	62	0	0	1	1	1	8	3	1	1	0	2	sui	Sui MV Exh
1978	397	58	0	0	1	0	1	8	3	1	0	0	2	acc	Acc Non-coll MVA
1978	398	55	1	0	1	2	1	8	3	1	9	0	24	sui	Sui dom gas by pipe
1978	399	53	0	0	2	3	0	8	3	1	1	0	8	acc	Acc MV Exh
1978	400	43	0	0	1	0	1	8	3	1	1	0	1	sui	Sui MV Exh
1978	401	37	0	0	1	0	0	8	3	0	1	1	1	sui	Sui MV Exh
1978	402	24	0	0	3	0	0	8	3	1	1	1	5	sui	Sui MV Exh
1978	403	23	0	0	0	3	0	8	0	1	1	0	3	acc	Acc CO
1978	404	18	0	0	0	0	6	8	3	1	1	0	0	sui	Sui MV Exh
1978	405	4	0	0	0	0	0	8	3	1	1	1	0	acc	Acc confl CO
1979	406	63	1	0	2	0	0	8	3	1	1	0	24	acc	Acc confl CO
1979	407	61	0	1	3	0	0	8	0	1	1	0	8	acc	Acc Confl sm/fumes
1979	408	52	1	0	1	1	1	8	3	1	1	0	24	sui	Sui MV Exh
1979	409	51	0	1	3	0	0	8	3	1	1	0	8	acc	Acc Confl sm/fumes
1979	410	29	0	0	1	0	1	8	3	1	1	0	3	sui	Sui MV Exh
1979	411	28	0	0	1	0	2	8	3	1	1	1	8	acc	Acc confl CO
1979	412	23	1	0	1	2	6	8	3	1	1	1	4	acc	Acc confl CO
1979	413	22	1	0	0	0	0	8	3	1	1	0	0	sui	Sui Suff
1979	414	4	1	0	0	2	0	8	3	1	1	1	0	acc	Acc confl CO
1979	415	3	0	0	0	2	0	8	3	1	1	1	0	acc	Acc confl CO
1980	416	42	0	1	1	0	0	0	3	0	1	0	2	acc	Acc CO
1980	417	41	0	0	3	3	0	0	0	1	1	0	3	sui	Sui MV Exh
1980	418	37	0	4	3	0	1	8	3	1	1	1	0	sui	Sui MV Exh
1980	419	35	1	0	1	0	0	1	3	1	1	1	24	acc	Acc confl CO
1980	420	33	0	1	1	0	0	8	3	1	1	0	6	acc	Acc confl CO
1980	421	33	0	0	0	0	1	0	3	0	1	0	4	sui	Sui dom gas other
1980	422	32	0	0	1	2	0	1	0	0	1	1	5	und	UND burns/fire
1980	423	28	0	0	1	2	1	0	3	1	1	0	1	sui	Sui MV Exh
1980	424	27	1	0	0	0	0	1	0	1	1	1	24	acc	Acc confl CO
1980	425	22	1	0	0	0	0	8	3	1	1	1	24	acc	Acc confl CO
1980	426	14	0	0	0	0	0	1	3	1	1	1	0	acc	Acc confl CO
1980	427	2	1	0	0	0	0	8	3	1	1	1	0	acc	Acc confl CO
1980	428	1	1	0	0	0	0	1	3	1	1	1	0	acc	Acc util other CO
1980	429	0	1	0	0	0	0	8	3	1	1	1	0	acc	Acc confl CO
1981	430	70	0	0	2	2	0	0	3	0	1	0	5	acc	Acc CO
1981	431	51	0	1	3	0	0	0	3	0	1	0	9	acc	Acc confl CO
1981	432	47	1	0	1	0	0	1	3	0	1	1	9	acc	Acc MV Exh
1981	433	46	0	0	2	1	0	0	3	0	1	0	2	sui	Sui MV Exh
1981	434	41	1	0	1	1	0	1	3	0	1	0	0	sui	Sui MV Exh
1981	435	36	1	0	2	0	0	0	3	0	9	1	24	acc	Acc confl CO
1981	436	29	0	0	0	0	0	0	3	1	1	0	3	sui	Sui MV Exh
1981	437	22	0	0	0	0	0	1	3	0	1	1	6	acc	Acc util CO dom fuels
1981	438	18	0	0	0	0	0	1	3	0	9	1	5	acc	Acc MV Exh
1982	439	74	1	0	2	0	0	0	2	1	1	0	4	sui	Sui MV Exh
1982	440	70	0	0	2	0	0	1	2	1	1	1	8	sui	Sui MV Exh

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1982	441	50	0	0	1	0	0	0	0	1	1	0	5	acc	Acc MVA w/ CO
1982	442	46	0	0	1	0	0	0	2	0	9	0	3	sui	Sui MV Exh
1982	443	43	0	0	1	0	1	0	2	0	1	0	9	sui	Sui MV Exh
1982	444	40	1	0	3	0	1	0	2	0	1	0	2	acc	Acc confl CO
1982	445	33	0	0	0	0	0	0	3	0	1	0	3	acc	Acc confl CO
1982	446	32	1	0	1	0	1	0	2	1	1	0	24	sui	Sui Other CO
1982	447	31	0	0	1	0	0	0	2	1	0	0	5	sui	Sui MV Exh
1982	448	26	0	0	1	0	1	0	2	0	1	0	9	sui	Sui MV Exh
1983	449	59	1	0	1	0	1	0	2	1	9	0	24	sui	Sui MV Exh
1983	450	58	0	0	1	0	0	0	2	1	1	0	4	sui	Sui Other CO
1983	451	48	0	0	3	0	4	3	2	0	1	1	1	sui	Sui MV Exh
1983	452	39	0	0	1	1	0	0	3	0	1	0	42	sui	Sui MV Exh
1983	453	27	1	0	3	2	0	0	2	0	1	0	24	acc	Acc confl CO
1983	454	25	0	0	0	0	0	0	0	0	9	0	5	acc	Acc MV Exh
1983	455	24	0	0	3	2	1	0	2	1	1	0	9	sui	Sui MV Exh
1983	456	24	0	0	0	4	1	0	2	0	9	0	43	sui	Sui MV Exh
1983	457	22	1	3	0	2	6	0	2	1	1	0	0	sul	Sui MV Exh
1983	458	20	0	0	1	0	0	1	3	0	1	1	5	acc	Acc Non-coll MVA
1983	459	1	1	1	0	0	0	0	3	0	9	0	0	acc	Acc confl CO
1984	460	78	1	0	2	2	0	0	2	1	1	0	2	sui	Sui MV Exh
1984	461	77	0	0	1	0	2	1	2	1	1	1	12	acc	Acc util unsp CO
1984	462	68	0	0	1	0	1	0	2	1	1	0	9	sui	Sui MV Exh
1984	463	60	0	0	1	0	1	0	2	0	1	0	98	sul	Sui MV Exh
1984	464	59	1	0	1	1	1	0	0	0	1	0	24	sui	Sui MV Exh
1984	465	44	0	0	3	0	2	1	2	0	0	1	9	acc	Acc MV Exh
1984	466	37	0	0	1	0	1	0	0	0	1	0	5	sui	Sui MV Exh
1984	467	32	1	0	1	2	0	0	2	0	1	0	4	sui	Sui MV Exh
1984	468	31	0	0	0	0	0	1	3	0	1	1	9	acc	Acc Non-coll MVA
1984	469	31	0	0	3	0	0	0	2	0	1	0	5	sul	Sui MV Exh
1984	470	26	0	0	0	0	6	0	2	1	1	0	0	sui	Sui dom gas by pipe
1985	471	76	0	0	2	0	0	0	2	1	1	0	8	sui	Sui Other CO
1985	472	72	0	1	2	0	0	0	2	0	1	0	45	acc	Acc MV Exh
1985	473	62	1	0	2	0	1	0	2	0	1	0	1	sui	Sui Other CO
1985	474	53	1	0	1	0	0	0	2	0	1	0	4	sui	Sui MV Exh
1985	475	37	1	0	1	2	0	0	2	0	1	0	2	sui	Sui Other CO
1985	476	37	0	0	1	2	0	0	2	0	1	0	1	sui	Sui Other CO
1985	477	36	0	0	1	2	0	1	2	0	1	1	5	sui	Sui Other CO
1985	478	36	0	0	3	0	0	0	2	0	1	0	7	sui	Sui MV Exh
1985	479	34	0	0	3	0	1	0	2	0	1	0	2	sui	Sui Other CO
1985	480	27	0	0	1	0	6	0	2	0	1	0	0	sui	Sui MV Exh
1985	481	26	0	0	3	2	0	0	2	0	1	0	5	acc	Acc util unsp CO
1985	482	24	1	0	3	2	0	0	2	0	1	0	24	acc	Acc util unsp CO
1985	483	22	0	0	0	3	0	0	2	0	1	0	0	sui	Sui MV Exh
1986	484	73	0	0	1	0	0	1	0	1	2	0	99	acc	Conflagr private dwell
1986	485	67	1	0	2	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1986	486	66	0	0	2	0	0	0	2	2	2	0	99	sui	Sui MV Exh
1986	487	60	1	0	1	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1986	488	59	0	0	1	0	0	0	0	1	2	0	99	sui	Sui MV Exh
1986	489	36	0	0	3	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1986	490	34	0	0	3	0	0	0	2	1	2	0	99	sui	Sui MV Exh

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1986	491	26	0	0	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1986	492	26	0	1	0	0	0	0	2	1	2	0	99	sui	Sui CO
1986	493	24	0	0	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1986	494	20	0	0	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1987	495	48	1	0	3	0	0	0	2	2	2	0	99	sui	Sui CO
1987	496	35	1	0	1	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1987	497	34	0	0	0	0	0	0	0	1	2	0	99	sui	Sui CO
1987	498	34	0	0	1	0	0	0	2	1	2	0	99	sui	Sui CO
1987	499	30	1	0	0	0	0	0	2	2	2	0	99	sui	Sui MV Exh
1987	500	26	1	0	3	0	0	0	2	1	2	0	99	sui	Sui CO
1987	501	22	0	0	3	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1987	502	18	0	0	0	0	0	1	2	1	2	1	99	sui	Sui CO
1988	503	58	0	0	0	0	0	0	0	1	2	0	99	sui	Sui CO
1988	504	57	0	0	3	0	0	0	3	1	2	0	99	sui	Sui MV Exh
1988	505	44	0	0	1	0	0	0	3	1	2	0	99	sui	Sui CO
1988	506	38	0	0	1	0	0	0	3	1	2	0	99	sui	Sui MV Exh
1988	507	36	0	0	1	0	0	0	0	1	2	0	99	sui	Sui CO
1988	508	31	0	0	3	0	0	0	3	1	9	0	99	sui	Sui MV Exh
1988	509	22	0	0	0	0	0	0	0	1	2	0	99	acc	Acc util & CO
1988	510	20	1	0	0	0	0	0	0	1	2	0	99	sui	Sui MV Exh
1988	511	19	1	0	0	0	0	0	0	1	2	0	99	sui	Sui MV Exh
1988	512	18	1	0	0	0	0	0	0	1	2	0	99	sui	Sui CO
1989	513	96	0	0	9	0	0	0	2	1	2	0	99	acc	acc burns
1989	514	76	1	0	1	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1989	515	68	0	0	1	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1989	516	64	0	0	1	0	0	1	2	1	2	1	99	acc	Conflagr private dwell
1989	517	63	0	0	1	0	0	0	7	1	2	0	99	acc	Acc util & CO
1989	518	50	0	0	1	0	0	2	2	1	2	1	99	sui	Sui MV Exh
1989	519	43	0	0	3	0	0	1	2	1	2	1	99	acc	Acc CO fr dom fuel
1989	520	42	0	1	3	0	0	0	2	1	2	0	99	acc	Acc MV Exh
1989	521	39	0	0	1	0	0	0	3	2	2	0	99	sui	Sui MV Exh
1989	522	32	1	0	1	0	0	0	2	1	2	0	99	sui	Sui CO
1989	523	24	1	1	0	0	0	0	2	1	2	0	99	acc	Acc MV Exh
1989	524	22	0	0	0	0	0	0	2	1	2	0	99	acc	Acc CO unsp
1989	525	19	0	3	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1989	526	19	0	3	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1989	527	18	1	0	0	0	0	1	2	1	2	1	99	sui	Sui CO
1989	528	17	0	0	0	0	0	0	0	1	2	0	99	sui	Sui Hanging
1989	529	17	0	0	0	0	0	0	0	1	2	0	99	sui	Sui MV Exh
1990	530	66	0	0	3	0	0	0	2	2	9	0	99	acc	Cardiac !
1990	531	63	1	0	1	0	0	0	2	1	2	0	99	sui	Sui CO
1990	532	42	0	0	1	0	0	1	2	1	9	1	99	acc	Other ill-def/unk cause
1990	533	36	0	0	1	0	0	0	2	2	9	0	99	sui	Sui MV Exh
1990	534	34	0	0	3	0	0	0	2	1	9	0	99	sui	Other ill-def/unk cause
1990	535	24	0	0	1	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1990	536	22	0	0	0	0	0	1	2	1	2	1	99	acc	Acc CO unsp
1991	537	49	0	0	1	0	0	1	2	1	2	1	99	acc	Conflagr private dwell
1991	538	48	1	0	3	0	0	1	2	1	2	1	99	sui	Sui CO
1991	539	42	1	0	1	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1991	540	41	0	0	1	0	0	0	2	1	2	0	99	sui	Sui MV Exh

yod	ID#	age	sex	race	mar	res	pob	sp	pod	aut	inj	ss	occ	mod	ucd**
1991	541	39	0	0	1	0	0	0	0	1	2	0	99	sui	Sui MV Exh
1991	542	37	0	0	3	0	0	0	2	1	2	0	99	sui	Sui CO
1991	543	33	1	0	3	0	0	1	0	1	2	1	99	acc	Conflagr private dwell
1991	544	33	0	0	1	0	0	0	0	1	2	0	99	sui	Sui CO
1991	545	27	1	0	3	0	0	0	2	1	2	0	99	sui	Sui CO
1991	546	25	0	0	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1991	547	24	0	1	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1991	548	24	1	0	0	0	0	0	2	1	2	0	99	sui	Sui CO
1991	549	4	1	0	0	0	0	1	0	1	2	1	99	acc	Conflagr private dwell
1992	550	77	0	0	1	0	0	0	2	1	2	0	99	sui	Other ill-def/unk cause
1992	551	68	1	0	2	0	0	2	0	1	2	1	99	und	Pois hormone or sub
1992	552	55	0	0	1	0	0	1	2	1	2	1	99	sui	Other ill-def/unk cause
1992	553	43	1	0	1	0	0	0	0	1	2	0	99	sui	Other ill-def/unk cause
1992	554	36	1	0	3	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1992	555	33	0	0	0	0	0	0	2	2	2	0	99	sui	Sui MV Exh
1992	556	27	0	0	0	0	0	0	2	1	2	0	99	sui	Other ill-def/unk cause
1992	557	21	0	0	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1993	558	76	0	0	3	0	0	0	2	1	9	0	99	acc	Conflagr private dwell
1993	559	59	0	0	1	0	0	1	2	1	9	1	99	acc	Conflagr private dwell
1993	560	55	0	0	1	0	0	1	2	1	9	1	99	acc	Acc CO unsp
1993	561	49	0	0	0	0	0	0	2	2	2	0	99	sui	Sui CO
1993	562	47	1	3	2	0	0	0	2	1	9	0	99	acc	Acc CO unsp
1993	563	42	0	0	1	0	0	0	2	1	1	0	99	sui	Sui CO
1993	564	38	0	0	9	0	0	0	2	1	9	0	99	sui	Sui MV Exh
1993	565	34	0	0	1	0	0	1	2	1	2	1	99	acc	Acc CO unsp
1993	566	34	0	0	0	0	0	0	2	1	9	0	99	sui	Sui MV Exh
1993	567	33	0	0	1	0	0	0	2	1	9	0	99	und	UND
1993	568	28	0	0	1	0	0	0	0	1	2	0	99	sui	Sui Suff
1993	569	24	0	0	0	0	0	0	2	1	9	0	99	acc	Acc CO unsp
1993	570	21	0	0	0	0	0	1	2	1	9	1	99	sui	Sui CO
1993	571	21	0	0	1	0	0	1	2	1	2	1	99	acc	Acc CO unsp
1994	572	63	0	0	1	0	0	0	2	1	2	0	99	sui	Sui CO
1994	573	49	0	0	1	0	0	1	2	2	2	1	99	sui	Sui CO
1994	574	39	0	0	0	0	0	0	0	2	9	0	99	sui	Sui CO
1994	575	39	0	0	0	0	0	0	0	1	2	0	99	sui	Sui CO
1994	576	32	0	0	1	0	0	1	2	1	2	1	99	sui	Sui MV Exh
1994	577	30	0	0	0	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1995	578	61	1	0	3	0	0	0	2	1	9	0	99	sui	Sui CO
1995	579	57	0	0	1	0	0	0	2	1	2	0	99	sui	Sui CO
1995	580	55	0	0	3	0	0	1	2	1	2	1	99	sui	Sui CO
1995	581	53	0	0	1	0	0	0	2	1	2	0	99	sui	Sui CO
1995	582	51	0	0	1	0	0	0	2	2	2	0	99	sui	Sui CO
1995	583	50	1	0	3	0	0	0	2	1	1	0	99	sui	Sui CO
1995	584	47	1	0	1	0	0	0	2	1	2	0	99	sui	Sui CO
1995	585	46	1	0	3	0	0	0	2	2	2	0	99	sui	Sui CO
1995	586	46	0	0	3	0	0	0	2	1	2	0	99	sui	Sui MV Exh
1995	587	44	0	0	1	0	0	0	0	1	2	0	99	sui	Sui CO
1995	588	37	0	0	1	0	0	0	2	1	9	0	99	acc	Acc CO unsp

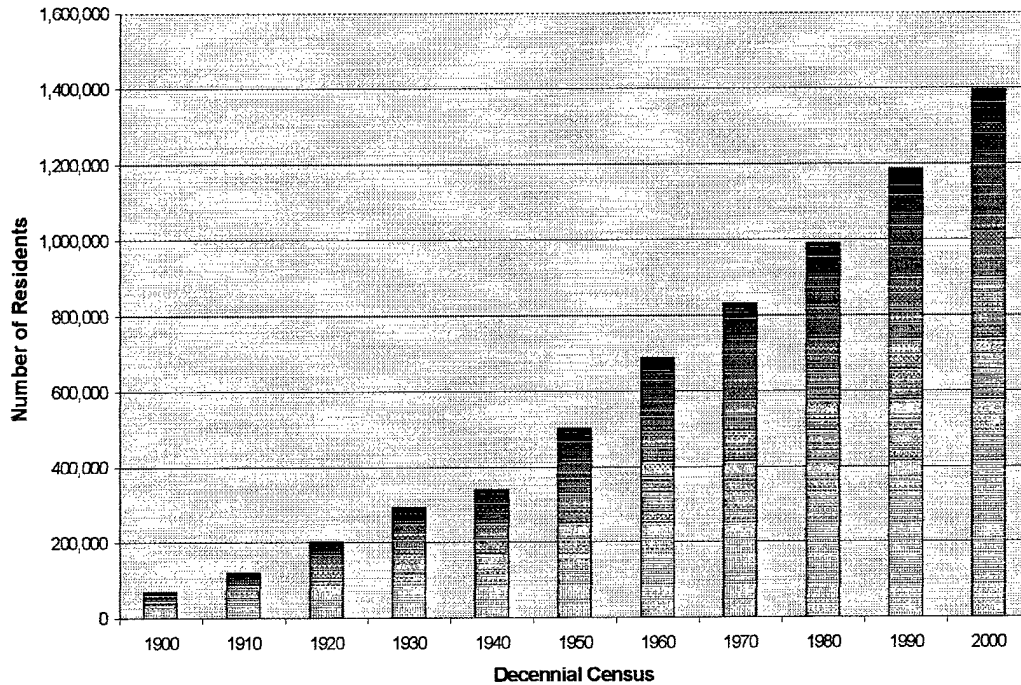
APPENDIX I

Database variable codes and descriptions

yod	Year of death	(self-explanatory)	
ID #	Identifier assigned for this study	1 through 588 (<i>assigned chronologically by author</i>)	
age	Age (in years) at time of death	(self-explanatory)	
sex	Gender	0 = Male	1 = Female
race	Race	0 = White	1 = Black
		2 = Native American	3 = Asian
		4 = Other	7 = Unknown
mar	Marital status	0 = Never married	1 = Married
		2 = Widowed	3 = Divorced
		7 = Unknown	9 = Blank on certificate
res	Residence	0 = San Antonio	1 = Other Bexar County city
		2 = Rural Bexar County	3 = Other Texas county
		4 = Other U.S. state	5 = Mexico
		6 = Other foreign country	7 = Unknown
		9 = Blank on certificate	
pob	Place of birth	0 = Texas	1 = Other U.S. state
		2 = Mexico	3 = Puerto Rico
		4 = Cuba	5 = Other Hispanic country
		6 = Other country	7 = Unknown
sp	Spanish origin	0 = Not Spanish	1 = Mexican
		2 = Puerto Rican	3 = Cuban
		4 = Other Spanish	7 = Unknown
		8 = Not asked (<i>prior to 1980</i>)	9 = Blank on certificate
pod	Place of Death	0 = Hospital	1 = Other institution
		2 = Other place	3 = "Dead on arrival"
		7 = Unknown	9 = Blank on certificate
aut	Autopsy performed	0 = Yes	1 = No
		2 = ? (<i>not defined in scheme!</i>)	7 = Unknown
		8 = Not asked (<i>in 1940-1948</i>)	9 = Blank on certificate
inj	Death due to occupational injury	0 = Yes	1 = No
		2 = ? (<i>not defined in scheme!</i>)	7 = Unknown
		9 = Blank on certificate	
ss	Spanish surname identifier	0 = Not a Spanish surname	1 = Spanish surname
occ	Occupation (1970 Census definition)	0 = No occupation	1 = Professional & Technical
		2 = Managers & Admin.	3 = Sales
		4 = Clerical	5 = Craft trades
		6 = Operatives (ex. Transport)	7 = Transport Equipment Op.
		8 = Laborer (ex. Farm)	9 = Service (ex. private household)
		11 = Farmers, Farm Managers	21 = Private household
		22 = "Domestic"	23 = "Housework"
		24 = Housewife, househusband	41 = Air Force
		42 = Army	43 = Navy
		44 = Marine Corps	45 = Military
		72 = Maintenance, general	97 = Unemployed
		98 = Uncodeable	99 = Blank on certificate
mod	Manner of death (<i>coded by author</i>)	acc = Accident	sui = Suicide
		hom = Homicide (<i>none found</i>)	und = Undetermined
ucd	Underlying cause of death	Listed in original database as ICD-9 codes; converted here to abbreviated descriptions. (See also APPENDIX G.)	

APPENDIX J

Population changes in Bexar County, Texas, 1900-2000*



<u>Census</u>	<u>Population</u>
1900	69,422
1910	119,676
1920	202,096
1930	292,533
1940	338,176
1950	500,460
1960	687,151
1970	830,460
1980	988,800
1990	1,185,394
2000	1,392,931

* Source: U.S. Bureau of the Census – <http://www.census.gov/population/cencounts/tx190090.txt>

APPENDIX K

Estimates of 1990 Bexar County population by age, sex, race, and Hispanic ethnicity*

Sex / Race / Ethnicity	Age Groups						Totals**
	0-9	10-19	20-29	30-39	40-49	50-59	
White Non-Hispanic Males	33,001	32,431	40,201	42,137	33,834	23,096	242,047
White Non-Hispanic Females	31,598	29,675	39,722	43,388	34,112	23,365	254,730
White Hispanic Males	60,032	55,172	51,649	44,633	29,216	17,988	283,227
White Hispanic Females	57,735	53,792	52,657	46,965	33,004	21,825	300,319
Black Males	8,069	7,387	7,637	6,997	4,372	3,080	41,544
Black Females	7,838	7,067	7,795	7,867	4,773	3,446	44,682
Am. Indian & Alaska Native Males	404	386	440	429	311	168	2,350
Am. Indian & Alaska Native Females	394	387	424	400	291	171	2,291
Asian & Pacific Islander Males	1,163	1,336	1,543	1,261	765	425	6,931
Asian & Pacific Islander Females	1,172	1,345	1,678	2,057	1,538	943	9,462
Totals	201,406	186,978	203,746	195,924	142,216	94,507	1,187,593
Hispanic Males, All Races	61,144	56,065	52,516	45,325	29,608	18,225	287,665
Hispanic Females, All Races	58,809	54,644	53,567	47,688	33,430	22,113	304,916
Total Hisp.	119,953	110,709	106,083	93,013	63,038	40,338	592,581
Non-Hispanic Males, All Races	41,525	40,647	48,954	50,132	38,890	26,532	288,434
Non-Hispanic Females, All Races	39,928	37,622	48,709	52,779	40,288	27,637	306,578
Total Non-Hisp.	81,453	78,269	97,663	102,911	79,178	54,169	595,012
Total Males, All Races	102,669	96,712	101,470	95,457	68,498	44,757	576,099
Total Females, All Races	98,737	92,266	102,276	100,467	73,718	49,750	611,494
Totals	201,406	188,978	203,746	195,924	142,216	94,507	1,187,593

* Source: U.S. Bureau of the Census -- http://leire.census.gov/popest/archives/county/co_casfrn.php

** These totals do not precisely match those shown in APPENDIX J because these data are Census Bureau estimates rather than actual census counts. Use of these estimates was necessary because actual 1990 census figures, based on self-reporting, included a large number of "Other" responses as to race (17.1% of the total population). It became evident that this was caused by about one-third of the county's white ethnic Hispanics, who had opted not to report themselves as racially "white."

APPENDIX L

Review of the 9 CO-related deaths with manner of death being "undetermined"

ID	Year of Death	Demographics & Listed Causes of Death
209	1966	46 y/o married non-hispanic white male resided in a Bexar City other than San Antonio. Occupation listed as "military." E 982.0 Poisoning by other gas, undetermined whether accidental or purposely inflicted. Motor vehicle exhaust gas. 514.0 Pulmonary congestion & hypostasis. 986.0 Toxic effect of carbon monoxide. (From all sources.)
249	1968	80 y/o widowed non-hispanic white female resided in San Antonio; former "professional/technical" career. E 982.0 Poisoning by other gas, undetermined whether accidental or purposely inflicted. Motor vehicle exhaust gas. 986.0 Toxic effect of carbon monoxide. (From all sources.)
291	1970	23 y/o married hispanic white male, born in Mexico & resided in San Antonio. Worked as an equipment operator. 242.9 Thyrotoxicosis. 427.5 Cardiac dysrhythmias. Cardiac arrest. 852.0 Subarachnoid, subdural, & extradural hemorrhage following injury. Without mention of intracranial wound. 864.0 Injury to liver. Without mention of open wound into cavity. E 982.0 Poisoning by other gas, undetermined whether accidental or purposely inflicted. Motor vehicle exhaust gas.
369	1975	44 y/o divorced non-hispanic white male craftsman, resided in San Antonio. E 982.0 Poisoning by other gas, undetermined whether accidental or purposely inflicted. Motor vehicle exhaust gas. 305.0 Non-dependent use of drugs. Alcohol. 986.0 Toxic effect of carbon monoxide. (From all sources.)
374	1976	39 y/o married non-hispanic white male. Air Force; resided in San Antonio. E 982.0 Poisoning by other gas, undetermined whether accidental or purposely inflicted. Motor vehicle exhaust gas. 986.0 Toxic effect of carbon monoxide. (From all sources.)
376	1976	36 y/o non-hispanic black female of unrecorded marital status, resided in San Antonio. Career coded as "professional/technical." E 982.0 Poisoning by other gas, undetermined whether accidental or purposely inflicted. Motor vehicle exhaust gas. 986.0 Toxic effect of carbon monoxide. (From all sources.)
422	1980	32 y/o married hispanic (Mexican) white male craftsman, resided in rural Bexar County. E 988.1 Injury by other & unspecified means, undetermined whether accidental or purposely inflicted. Burns, fire. 948.6 Burns classified according to extent of body surface involved. 60-69%. 986.0 Toxic effect of carbon monoxide. (From all sources.)
551	1992	68 y/o widowed hispanic (Puerto Rican) white female. Resided in San Antonio; no occupation listed. 962.2 Poisoning by hormones & synthetic substitutes. Ovarian hormones & synthetic substitutes. 851.0 Cerebral laceration & contusion. Without mention of open intracranial wound. 854.0 Intracranial injury of other & unspecified nature. Without mention of open intracranial wound. 986.0 Toxic effect of carbon monoxide. (From all sources.)
567	1993	33 y/o married non-hispanic white male. Resided in San Antonio; no occupation listed. E 982.1 Toxic effect of solvents other than petroleum-based. Carbon tetrachloride. 986.0 Toxic effect of carbon monoxide. (From all sources.)

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VITA

Lawrence Todd Burd was born in Latrobe, Pennsylvania [REDACTED], the eldest of three sons of [REDACTED]. He graduated with high honors from Derry Area Senior High School in 1977, and continued studies at the Pennsylvania State University on a four-year Air Force ROTC scholarship. In 1981 he graduated with distinction from Penn State with a Bachelor of Science degree in Biochemistry, and entered active duty a few months later as a communications-electronics officer. In 1986 he became a special agent in the Air Force Office of Special Investigations (AFOSI). He completed a Master of Forensic Sciences degree at George Washington University in 1989, and later that year finished a Master of Science in Administration through Central Michigan University. He was assigned as the Regional Forensic Consultant for AFOSI's Northeastern Region at Wright-Patterson Air Force Base, Ohio, and later served in the same role for the Pacific Region during a one-year tour at Osan Air Base, South Korea. After being selected for an Air Force Health Professions Scholarship, he left active duty in 1992 to attend the Pennsylvania State University's College of Medicine. Following graduation in 1996, Dr. Burd completed an intern year at Wilford Hall Medical Center, Lackland Air Force Base, Texas. He continued there for an additional year of residency training in psychiatry before opting to pursue flight medicine. From 1998 to 2000 he was assigned to an Aerial Refueling Squadron at McGuire Air Force Base, New Jersey. While there, he deployed to Germany during early tensions in the Balkans, and deployed to the Middle East three times in support of Operation SOUTHERN WATCH. He later served as Commander of Flight Medicine in the 305th Medical Group before leaving New Jersey in 2001. Returning to Texas, he began a Master of Public Health degree at the San Antonio campus of the University of Texas Health Science Center at Houston as part of a residency in aerospace medicine. He is married to [REDACTED] of [REDACTED] Pennsylvania, and they have a daughter, [REDACTED] who turns 11 in August 2002.

This thesis was typed by Lawrence T. Burd.